

ELECTROCARDIOGRAPHY AND CARDIAC PATHOLOGY

with Especial Reference to Ventricular Preponderance.

A Study Comparing Electrocardiographic and Clinical
Observations with Gross and Microscopical
Postmortem Findings.

A DISSERTATION

submitted in partial fulfillment of the requirements
for the degree of
Doctor of Philosophy,
in the Graduate School of the University of Michigan
by
George R. Herrmann

Diss. Ann Arbor.

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A Study Comparing Electrocardiographic and Clinical Observations with Gross and Microscopical Postmortem Findings.

by

GEORGE R. HERRMANN

The introduction of every new instrument of precision for the study of medical problems is followed by the production of a mass of literature. A portion of this matter is not infrequently made up of insufficiently controlled data and hasty conclusions. The failure of the method to fulfill the expectations based on such more or less inferential deductions, may lead to much undue criticism. Electrocardiography has suffered to some extent in this country from this very unfortunate practice. The physiological principles of electrocardiography were distinctly advanced by the inventor of the method, the Dutch physiologist, Einthoven. Others, as Lewis, Eppinger, Rothberger, etc., and their associates have made important contributions especially to the physiological, but to a definite extent also to the pathological phases of the science. Some Americans have made careful observations, but some of our literature is confusing and unreliable because of the great proportion of theory as contrasted to the paucity of reliable experimental and pathological data.

This study was undertaken to establish, by a large series of cases, the actual status of the electrocardiographic signs of preponderance, and at the same time to attempt to correlate whatever microscopical findings were present in the myocardium of each case with the form of the electrocardiogram that was obtained during the life of the patient.

HISTORICAL

When Einthoven (3, 4) began to apply the string galvanometer to the study of heart disease, he observed that certain valve lesions, when accompanied by great cardiac enlargement, yielded electrocardiograms of distinctive type. In cases of mitral or pulmonary stenosis in which preponderant hypertrophy of the right ventricle was believed to be present, the tallest QRS-summit occurred in lead III and the greatest downward deflection in lead I (Fig.1A). In aortic insufficiency and in other conditions in which preponderant hypertrophy of the left ventricle appeared to be present, the electrocardiographic picture, when compared with that of mitral disease, was inverted; the tallest summit occurred in lead I, the deepest deflection in lead III (Fig.1B). Einthoven also noted that these abnormal curves were often of great amplitude.

Einthoven did not attempt to give a complete explanation of the mechanism through which hypertrophy of the one or the other ventricle produces characteristic changes in the form of the electrocardiogram. He appears to have believ-

ed these changes due to some peculiarity of the spread of the excitation process such that in left ventricular hypertrophy, the first region activated, or the region of greatest initial activity, lay near the cardiac apex, while in right ventricular hypertrophy, this region lay more toward the ventricular base. Whether he believed that unilateral hypertrophy was accompanied by modifications of the conducting system itself, or whether he believed only that it altered the positional relation of the ventricular regions which normally received the impulse earliest to the muscle mass as a whole, is not clear.

Einthoven's observations have been confirmed repeatedly and extended. We need refer here only to the work of Lewis (9) which may be briefly summarized as follows:

1—In aortic insufficiency, Einthoven's signs of left ventricular hypertrophy are usually, but not invariably, found. In rare instances the electrocardiogram may suggest right ventricular hypertrophy rather than left. The average values of the individual electrocardiographic deflections in a large series of cases show, as compared with the average values for normal subjects, a conspicuous increase in the amplitude of R1 (R in lead 1) and S3 and a decrease in amplitude of S1 and R3; that is, the average curve exhibits, in some measure, the signs of preponderant hypertrophy of the left ventricle.

2—In mitral stenosis, Einthoven's signs of right ventricular hypertrophy are the rule and are shown, in some degree, by the average curve. Cases which fail to follow the rule, though less frequent than in aortic insufficiency, are not uncommon.

3—In congenital heart disease, the average values of the various deflections indicate a much greater degree of right ventricular preponderance than do the corresponding average values in mitral stenosis. It is in congenital pulmonary stenosis that Einthoven's signs of right ventricular hypertrophy are most fully developed.

4—The electrocardiogram of the new-born infant shows the signs of right ventricular preponderance almost invariably. These disappear gradually and, between the third and fourth months of life, the curve approaches the form seen in the normal adult.

5—Many normal electrocardiograms exhibit Einthoven's signs of right or of left ventricular hypertrophy in minor degree, and normal electrocardiograms may be divided into two classes according as they conform more to the former type or to the latter.

These observations tended to confirm Einthoven's contention that unilateral hypertrophy produces characteristic electrocardiographic signs. It was necessary, however, to explain the absence of these signs in many cases of aortic insufficiency and of mitral stenosis; for it was believed that

unilateral hypertrophy was almost invariably present in these disorders; and to explain their presence in the new-born and in certain normal adults. Lewis sought the explanation of these difficulties in a study of the relative weight of the two ventricles under various circumstances. He devised a method of sectioning the heart which permits the muscle of each ventricle and that of the septum to be weighed separately. The ratio L-R may then be determined by dividing the weight of the left ventricle by that of the right, the weight of the septal muscle being neglected. When this method had been carried out in a large series of cases, it was found that although preponderant hypertrophy of the left ventricle is the rule in aortic insufficiency, and preponderant hypertrophy of the right in mitral stenosis, numerous exceptions occur. The absence of the expected electrocardiographic signs in certain patients with these valve lesions thereby became intelligible. It was also found that the average value of the ratio L-R was higher in aortic insufficiency and lower in mitral stenosis than in a series of controls; this seemed to explain the form of the average curve in each type of case. The ratio L-R of the controls varied within wide limits and the occurrence of signs of slight right or slight left preponderance in normal individuals could be attributed to unusual variations in this ratio. And lastly, Müller, in a similar study of the relative weight of the two ventricles, had found that the right ventricle of the new-born infant is considerably heavier in comparison with the left than is that of the adult, and that this condition persists until about the third month of life.

These results seemed to offer a complete explanation of the occurrence of the electrocardiographic signs in question and to place Einthoven's views upon a solid foundation. A more direct test of the value of these signs was, however, attempted. In nine cases the ratio L-R and the electrocardiographic signs of unilateral hypertrophy exhibited before death were compared. Six cases have been added to this series by Cotton (2). Except in two of these fifteen cases the electrocardiographic indications and the type and degree of preponderant hypertrophy present correspond remarkably well.

The following explanation offered by Lewis (8) for the occurrence of characteristic electrocardiograms in association with unilateral hypertrophy is based upon his interpretation of the normal ventricular complex. He has shown that the normal QRS group is produced by the algebraic summation of the left ventricular electrocardiogram (levocardiogram) and the right ventricular electrocardiogram (dextrocardiogram)*. The early phases of these curves have been record-

* (Note):—As Einthoven (4) has pointed out, the statement that the ventricular electrocardiogram is the result of a summation of right and left ventricular effects is equivalent to the assertion that the whole is equal to the sum of its parts. But the right and left ventricles, as opposed to the base and apex, for example, are natural subdivisions; each ventricle has its own conducting system and is, therefore, in some measure, capable of independent activity.

ed and the part that each plays in the formation of the normal QRS group has been established; Q1 and S3 are left ventricular effects; Q3 and S1 are right ventricular effects; R1 is chiefly a left and R3 chiefly a right ventricular effect. Lewis concluded, therefore, that the tall R1 and deep S3 of left ventricular hypertrophy are the result of preponderance of the levocardiogram, while the tall R3 and deep S1 of right ventricular hypertrophy are the expression of preponderance of the dextrocardiogram. He pointed out that, in accordance with this view, Q1 is large in left and Q3 in right ventricular preponderance. His demonstration that anticlockwise rotation of the electrical axis is common to both the levocardiogram and the curves of left ventricular preponderance, while the dextrocardiogram and right ventricular preponderance curves show clockwise rotation, also supports this conclusion.

The problem of explaining Einthoven's signs was thus reduced to the problem of explaining how preponderant hypertrophy of the one or the other ventricle gives rise to preponderance of the corresponding unilateral electrocardiogram. In the normal electrocardiogram the effects of the two ventricles are fairly well balanced in spite of the greater mass of the left; nevertheless, it seems not improbable that the amplitude of the electrical effects of either ventricle depends, in some measure at least, upon its mass. A relative increase in the mass of the one or the other chamber may destroy the balance between right and left ventricular effects and enable the corresponding unilateral electrocardiogram to dominate the combined curve.

METHODS

In collecting the material upon which this dissertation is based, we had in mind the addition of a considerable number of cases to the small series already reported (1) (2) and (9). This seemed desirable in order that the relation between the ratio L-R and Einthoven's signs might be more exactly determined. We have followed, for the most part, the methods used by the authors mentioned so that our data might be comparable to theirs. In order, however, to obtain a wider range of observations, and at the same time as many cases as possible, we have not restricted our attention to examples of cardiac disease. Electrocardiograms were made routinely in all cases in which cardiac abnormalities were known to be present or were suspected to exist. For the purposes of this study, electrocardiographic observations were also made upon all patients who were critically ill, whatever the primary disease might be. As a result, our series of cases differs from those of Lewis and Cotton in that it includes many cases in which hypertrophy of the heart was not present.

The electrocardiographic curves were standardized in the usual way and the effect of introducing a difference of po-

tential of one millivolt into the circuit containing the string and the patient was photographed in each instance. In measuring the curves, corrections were made for errors in standardization. When conspicuous overshooting was present, due to high skin resistance, the curves were discarded. In most of our cases only one series of curves were taken. In those instances in which curves were made on several occasions, only the final curve was measured, except in a few cases in which it differed conspicuously in form from the curves made previously.

METHOD OF PREPARING AND SECTIONING THE HEART

In order to allow a more complete examination of the heart at the time of the postmortem, it was necessary to make some minor alterations in the Lewis method of preparing the heart for sectioning.

The fresh heart freed of the parietal pericardium, with the large vessels cut short was received at the autopsy. After the organ was weighed, the auricles were removed, and the mitral, aortic, tricuspid and pulmonary valves were carefully examined and tested by the hydrostatic test. The mitral and tricuspid leaflets were then removed and the aorta and pulmonary artery were separated from the ventricular muscle. The valve leaflets, vessels, and auricular muscle were then returned to the pathologist for further examination.

The capacity of each ventricle was determined* by measuring the volume of mercury necessary to fill its cavity. Long pins were thrust through the ventricular base and with the mercury still in place, the heart was suspended in a vessel containing 10% formol. From this point onward the method of Lewis was strictly followed up to the time when the organ was sectioned.

Unfortunately, Lewis has described his method of sectioning the heart but briefly and we misunderstood his directions. Hence we have the origin of METHOD A. The first cut was made parallel to and along the right margin of the septum in such a way that the cavity of the right ventricle remained closed on its septal side by a very thin slice of septal tissue. The cut was begun posteriorly and was brought forward until it reached the conus tendon which was then followed as in the method of Lewis. On the anterior surface of the heart, the cut followed the interventricular sulcus. This cut was continued downward until the right ventricle was completely separated from the remainder of the heart muscle. The second cut was made tangential to the cavity of the left ventricle on its septal side, but instead of being carried straight through to the center of the conus tendon, (as in the LEWIS METHOD Fig. 2) its anterior part was curved to the

*(Note):—The ventricular capacities of the first twenty hearts obtained were not measured. These hearts were maintained in a distended condition by filling the ventricular cavities with cotton wool.

left so that it was maintained parallel to the first cut throughout its course (Fig. 3A). We did not discover our unplanned modification until the first 50 hearts of our series had been divided. The cuts, however, were such that Lewis' directions could also be directly applied, consequently these hearts were resectioned according to the method of Lewis, which was followed as the final step in all cases.

We felt, especially with the first method employed, some uncertainty in making the cuts, and sought some way in which this might be avoided. The following method (OUR METHOD B Fig. 3B and 4A, B & C) of separating the right and left ventricles seems to us less arbitrary and less subject to error than those heretofore employed. The method not only gives satisfactory results in itself, but also may be used as an important step in the Lewis method, making the latter more limited and more accurate and at the same time quicker and less cumbersome. The distention of the ventricles is furthermore not so essential, as is the case in the original method.

The fixed ventricles are divided by cuts at right angles to the long axis of the heart into a series of sections, each about $1\frac{1}{2}$ cm. in thickness. If this is done with a sharp knife, a thin but clear-cut line, formed by the intersection of the scroll muscle layers, is seen running through the center of the septum in an anterior-posterior direction. This line is well shown in Fig. 4A, photographs of one of the sections of the heart in Cases 25 and 4. Each section is divided into right and left divisions by a cut which follows this line throughout its course; near the anterior and posterior surfaces of the surfaces of the heart where the line forks, the cut bisects the angle between the branches. The septal muscle is distributed by the cut between the two ventricles and is not considered separately, as is shown in Fig B & C.

This method (Method B) was employed in 29 cases in the first series and in 21 cases in a second series, making a total of 50 cases. The heart was sectioned in 21 instances by our first method (Method A) which was later discarded. All cases sectioned by methods A and B were later sectioned according to that of Lewis, and the latter method was employed some cases alone. The muscle weights obtained by these methods (A and B and Lewis') are given in Table 1; the muscle weights obtained are all included, the L-R ratio by each method estimated, the difference computed, and the averages calculated so that various methods used may be compared.

It will be seen that the ratio L-R determined by method B is practically invariably lower than that determined by the method of Lewis and also lower than that determined by the method A. The average difference between the ratio L-R determined by the Lewis method and the ratio L-R determined by method B is 0.162; the maximal difference is 0.68 and

the minimal difference-0.035. When the ratio L-R is high, the difference appears to be greater than when it is low; thus for 10 cases with L-R ratios ranging from 3.59 to 1.99 (Lewis' method) the average difference is 0.32; for 10 cases with ratios ranging from 1.97 to 1.80 it is 0.148; and for 10 cases with ratios ranging from 1.75 to 1.55 it is 0.17; for 10 cases from 1.56 to 1.42 it is 0.07 and for 10 cases from 1.41 to 0.95 it is 0.02. Assuming that the methods may be carried out with equal accuracy, the maximal error in the ratio L-R determined by either method will amount to approximately one-half the difference between the maximal (or minimal) and the average difference for a small range of L-R values. For our first group of cases (ratios between 3.59 and 1.96) the maximal difference is 0.68 and the average difference 0.36; the maximal error is, therefore, about 0.18. It appears from the table, however, that errors as great as this seldom occur.

Arranging the series according to L-R ratio (Lewis) into the four groups, used to get a more exact idea of the relation of the L-R ratio to the form of the electrocardiogram, we find the following: In group I with L-R ratios above 2.20, there are 6 comparisons with an average difference of 0.40 between Method B and the Lewis method. In group II L-R ratios 1.80 to 2.20 there are 14 comparisons with an average difference of 0.14. In group III L-R ratios 1.50 to 1.78, there are 16 comparisons with an average difference of 0.16. In group IV L-R ratios below 1.50, there are 14 comparisons with an average difference of 0.04.

It will be noted that when the hearts were sectioned by the Lewis method the average ventricular weight was about 18 grams less than when the first cuts were made in the first series, while in the second series (marked A, No. Z) the difference was practically nothing. The hearts of the first series had been stored in weak formol for some time and, although they were again placed in water for several days, they did not regain their original weights.

When the last cuts were made, in the first series, the thickness of the lateral wall of each ventricle at the junction of its basal and middle thirds was measured.

MICROSCOPICAL STUDY

After the gross weighing studies were completed, ten blocks of tissue about 1 cm. square and about .5 cm. thick were cut from the base, papillary muscle region, apex and upper septum of the ventricles. The blocks were dehydrated in 70%, 96%, and finally absolute alcohol, cleared in xylol and imbedded in paraffin in the routine way. Two ten micron sections were cut from each block, of which the one was stained with Van Gieson's and the other with the ordinary hematoxylin and eosin stains. The two sections from each block were mounted in balsam side by side on a glass

slide. In all, about 1200 sections were studied. The significant findings are noted in the last column of Table II of pathological finding, under the head of myocardial lesions. The complete descriptions of the gross and microscopical findings are included in each case history.

DESCRIPTION OF OBSERVATIONS

TABLES II, III, AND IV. Our observations are summarized in tabular form; the details of the clinical and postmortem observations are given in Table II. The cases are arranged in the order determined by the ratio L-R (Lewis method), beginning with the highest ratio and ending with the lowest. The term "heart weight" refers to the weight of the fresh heart as it was received from the pathologist. The figures for height and weight and the notes upon the condition of the kidneys and heart valves were abstracted from the pathological records; the blood pressure readings, from the clinical records. Notes relative to the condition of the cardiovascular system and to other matters of interest in relation to the position of the heart, etc. are recorded.

The electrocardiographic measurements are given in Table III; the age, ventricular weight, and ratio L-R are repeated here for comparison. The final column of the table contains the most important or the primary disease condition. Electrocardiographic measurements which fall outside the normal limits determined by Lewis and Gilder (10) are printed in (*italics*). The index was computed by subtracting the sum of R3 and S1 from the sum of R1 and S3. The normal range of the index is not given by Lewis and Gilder, but was determined from their tables; the maximal index in their series of 52 cases is 8.5; the minimal index is -14. Indices which fall outside these limits are printed in *italics*. Certain positive indices are followed by one or by two stars to indicate that the corresponding electrocardiograms show one (one star) or both (two stars) of the following signs of left ventricular preponderance: a—the tallest summit occurs in lead I, b—the deepest deflection occurs in lead III and exceeds the normal limit of 0.45 millivolt. Stars follow negative indices when the corresponding signs of right ventricular preponderance are present; the normal limit for S1 is 0.60 millivolt. We have adopted throughout the normal limits determined by Lewis and Gilder. The number of subjects studied by them was not large, but they were selected with great care.

In Table IV the average values of the electrocardiographic measurements and muscle weight determinations in various types of cases are given. Lewis' average values for controls, for aortic insufficiency, and for mitral stenosis are included for comparison.

THE NORMAL RANGE OF THE RATIO L-R AND OF THE VENTRICULAR-WEIGHT BODY-WEIGHT RATIO. In Lewis' series of "cancer controls" the ratio L-R ranges

from 1.46 to 2.06; in his series of "controls," from 1.57 to 2.18; in Cotton's series of controls, from 1.43 to 2.28, providing Case 10, an infant aged 2 days, be excluded. In our own series of controls (Table IV) it ranges from 1.46 to 2.14; or, if those cases in which an abnormal electrocardiogram was the chief cardio-vascular abnormality be excluded, from 1.46 to 1.96. Müller (12), who used a different and probably slightly less accurate method of sectioning the fresh heart, considered 0.400 the lower, and 0.650 the upper normal limit for the ratio R-L; these figures correspond to L-R ratios of 2.50 and 1.54 respectively. In all series of controls in which the Lewis method was used the average value of the ratio L-R lies in the immediate neighborhood of 1.80. For the purposes of this study, we consider ratios above 2.20 or below 1.50 abnormal.

In Table II we give for each case the ratio of the ventricular weight in grams to the body weight in grams. This ratio was computed also in all of the control cases published by Lewis. In his series of "cancer controls" it varies between 0.00200 and 0.00421 (average value 0.00301); in his series of "controls" it ranges from 0.00276 to 0.00480 (average value 0.00386). In our own series of controls it ranges from 0.00170 to 0.00390 (average value 0.00292). Müller's average values for this ratio range from 0.00371 to 0.00489. We believe that ratios above 0.00500 indicate definite ventricular hypertrophy.

COMPARISON OF THE RATIO L-R AND THE ELECTRO-CARDIOGRAPHIC SIGNS OF PREPONDERANCE OF THE ONE OR THE OTHER VENTRICLE.

Examination of Table II indicates that the relation between the form of the QRS group and the relative weight of the two ventricles is much less close than is, at present, generally believed. In order to get a more exact idea of this relation in our series of cases we have divided them, on the basis of the ratio L-R, into four groups:—

GROUP 1—L-R RATIO ABOVE 2.20; ABNORMAL LEFT VENTRICULAR PREPONDERANCE. Fifteen of our cases fall in this group. In only two of these does the final electrocardiogram indicate definite left ventricular preponderance (index positive—two stars). In four instances it indicates questionable left ventricular preponderance (index positive—one star). In five instances the final curve shows a tendency toward left ventricular preponderance (index positive—no star), and in an equal number of instances, a tendency toward right ventricular preponderance (negative index—no star). If we consider all the curves made, instead of the final curves alone, an abnormal degree of left ventricular preponderance might, perhaps, be suspected from the form of the electrocardiogram in eight cases. It is noteworthy that in five of these eight cases the ventricular weight ex-

ceeds 300 grams. Of the remaining seven cases in which the electrocardiogram shows no evidence of abnormal left ventricular preponderance the ventricular weight exceeds 300 grams in none; in only one does it exceed 200 grams.

GROUP II—L-R RATIO 1.80 TO 2.20, BOTH INCLUSIVE, LEFT VENTRICULAR PREPONDERANCE WITHIN NORMAL LIMITS. Sixteen of our cases fall in this group. The electrocardiogram indicates definite left ventricular preponderance in four cases, and questionable left ventricular preponderance in two. In three of these six cases the ventricular weight exceeds 250 grams. In eight cases the electrocardiogram indicates a tendency toward left ventricular preponderance; in one instance the index is zero. In one instance the electrocardiogram indicates questionable right ventricular preponderance. In none of these cases does the ventricular weight reach 200 grams.

GROUP III—L-R RATIO 1.50 TO 1.78 (INCLUSIVE), RIGHT VENTRICULAR PREPONDERANCE WITHIN NORMAL LIMITS. Twenty-one of our cases fall in this group. In one case the electrocardiogram indicates definite left ventricular preponderance; in one case it indicates definite right ventricular preponderance. In both of these cases the ventricular weight is low. In seven cases the electrocardiogram indicates a tendency toward left ventricular preponderance and in eleven cases, a tendency toward right ventricular preponderance; in one case the index is zero. In none of these nineteen cases does the ventricular weight reach 250 grams.

GROUP IV—L-R RATIO BELOW 1.50, ABNORMAL RIGHT VENTRICULAR PREPONDERANCE. In this group there are but seven cases; in none of these does the electrocardiogram indicate either definite or questionable right ventricular preponderance. In three cases it indicates a tendency toward right ventricular preponderance, and in an equal number of cases, a tendency toward left ventricular preponderance. In only one of these six cases does the ventricular weight exceed 250 grams. In one case the electrocardiogram indicates definite left ventricular preponderance; in this instance the ventricular weight is 199 grams.

To sum up in tabular form:

Group	No. of cases	Definite L. prepond.	Quest. L. prepond.	Tend. to L. prepond	Tend. to R. prepond	Quest. R. prepond.	Definite R. prepond.
I.	15(5)*	2(2)	6(3)	3(0)	4(0)	0(0)	0(0)
II.	16(3)	4(2)	1(1)	9(0)	0(0)	1(0)	0(0)
III.	21(0)	1(0)	0(0)	7(0)	11(0)	0(0)	1(0)
IV.	7(1)	1(0)	0(0)	3(0)	3(1)	0(0)	0(0)

There appears to be a distinct relation between the total ventricular weight and the frequency with which the electrocardiographic signs of preponderance occur. Of our 59 cases, 22 show L-R ratios outside the normal range. The signs of definite or questionable preponderance are found 5

*(Note):—The numbers in parentheses indicate the number of cases in which the ventricular weight exceeds 250 grams.

times in 6 instances in which the total ventricular weight exceeds 250 grams; they are found but 4 times in the 16 instances in which it falls below this figure. The signs of preponderance are, furthermore, much more reliable when the heart is greatly hypertrophied than when it is of relatively normal weight. The ratio L-R is above the normal average in every instance in which the signs of left ventricular preponderance are associated with a ventricular weight of over 250 grams; it is below the normal average in 2 of 7 cases in which these signs are associated with a ventricular weight of less than 250 grams. When the heart is of normal weight or is only slightly hypertrophied, there appears to be very little relation between the form of the electrocardiogram and the ratio L-R. The close relation which is found in the series of Lewis and Cotton is undoubtedly due to the fact that they dealt exclusively with greatly hypertrophied hearts; in only one of the fifteen cases published by them does the ventricular weight fall below 250 grams.

The relation between the ventricular weight and the occurrence of preponderance curves is well shown by the average values of Table IV. In all of those instances in which the average index indicates a conspicuous degree of right or left preponderance; in aortic insufficiency, in mitral stenosis, and in chronic hypertension; the average ventricular weight is high (250 grams or over), whereas in other groups of cases in which the average L-R ratio is equally abnormal but in which the average ventricular weight is low, the signs of preponderance are present in a much less evident degree.

DISCUSSION

The failure of a definite relation between the form of the electrocardiogram and the ratio L-R when the heart is of relatively normal weight; that is, when the ventricles weigh less than 250 grams, is of significance. The fact does not necessarily mean that we must abandon entirely our present conception of the manner in which preponderance curves arise. Furthermore, the apparent relation between the L-R ratio and the electrocardiogram when the heart is greatly hypertrophied need not be attributed to, as yet, unknown causes. The question arises as to whether the ratio L-R is not just one of several factors determining the form of the ventricular complex, a factor the influence of which increases with the increase of the ventricular weight. A brief consideration of the theory of the origin of preponderance curves and an examination of certain factors, which may disturb the relation between the L-R ratio and the form of the electrocardiogram, will furnish an answer to these questions.

RELATION OF THE MASS OF THE HEART TO THE AMPLITUDE AND DIRECTION OF ITS ELECTRICAL EFFECTS.

Our present conception of the origin of preponderance curves is based upon the fundamental assumption that there

is a relation between the mass of the heart muscle and the magnitude of the potential difference which it produces. This question of the existence of such a relation is a difficult one to answer; for the "manifest" potential difference at the body surface is a function of many variables.

It is true that the electrocardiogram of an infant does not differ materially in amplitude from that of an adult, although the heart of the latter is many times the more massive. But this may be due to the fact that the "manifest" potential difference decreases as the distance of the electrodes from the heart increases. We know also that the "manifest" potential difference must vary with the conductivity of the tissues that surround the heart and with the plane of the electrodes, and that variations in the amplitude of the electrical deflections may occur with variations in the contractility of the heart muscle (6). These and other similar factors tend to obscure any possible relation that may exist between the amplitude of the electrocardiogram and the weight of the heart. Since, however, most of them must affect the "manifest" potential differences produced by the two ventricles in approximately the same way and to the same degree*, they will not disturb a possible relation between the relative mass of these two chambers and the relative amplitude of their respective electrical effects.

Although the left ventricle is normally 1.8 times as heavy as the right, the two chambers produce electrical effects of approximately equal amplitude. If we hold that there is a relation between the mass of each ventricle and the amplitude of the corresponding unilateral electrocardiogram, we must admit that each gram of right ventricular muscle is equivalent, so far as its electrical effects are concerned, to 1.8 grams of left ventricular muscle. Such a relation is by no means as impossible as it sounds. The levocardiogram and the dextrocardiogram are resultants; each is produced by a combination of opposed forces and its amplitude is, therefore, a measure of the degree in which one group of forces overbalances the other. The distribution of the ventricular muscle may be such as to cause a greater lack of balance between the opposed forces of the right ventricle than between those of the left. It is possible also that the right ventricle is more advantageously placed than the left in that its major electrical axis (the electrical axis at the time when the difference of potential is at its maximum) falls more nearly in the plane

*(Note):—The influence of the conductivity of the tissues which surround the heart upon the form and amplitude of the electrocardiogram is difficult to estimate. If all tissues of the body conduct electrical currents equally well, then the "manifest" potential difference between two points at the body surface should vary inversely with the conductivity of the tissues. If on the other hand the conductivity of the heart muscle differs from that of the surrounding tissues, then the proportion of the total current that flows in the heart itself will vary with the relative conductivity of the cardiac tissue and the adjacent tissue. The left ventricle is surrounded by air-containing lung while the right ventricle lies adjacent to the liver and the anterior chest wall; it is possible that this may affect the relative amplitude of the effects of these chambers at the body surface.

of the three leads. And lastly, the thinness of the wall of the right ventricle compared to that of the left and possibly the arrangement of its conducting system may cause a greater concentration of its electrical effects in that, during a part of the QRS interval, a greater percentage of the right than of the left ventricular muscle is simultaneously passing into the active state. Finally, it should be pointed out that in the bird, in which the left ventricle is more than three times as heavy as the right, the levocardiogram dominates the bigram completely.

So far as we can see, then, there are no facts seriously in conflict with the belief that there is a relation between the mass of the one or the other ventricle and the amplitude of the corresponding unilateral electrocardiogram. There are, on the other hand, certain facts which suggest that such a relation does exist. The association of preponderance curves with aortic insufficiency, mitral stenosis, pulmonary stenosis, and chronic hypertension, and their occurrence in the newborn is, at present, difficult to explain on any other basis. Of equal importance is the fact that electrocardiograms of enormous amplitude often accompany great cardiac hypertrophy. The maximal deflection of the normal electrocardiogram seldom exceeds a value of 2 millivolts, whereas in pulmonary stenosis, deflections exceeding 4 millivolts are not infrequently encountered (Fig. 1). Deflections of this amplitude never occur, so far as we know, in the absence of great hypertrophy of the heart.

To sum up, we may say that the present day theory of preponderance curves gives a more simple and rational explanation of the known facts than any other that has been proposed and that there are no facts seriously in conflict with it.

THE INFLUENCE OF THE RELATIVE WEIGHT OF THE TWO VENTRICLES AND OF THEIR ABSOLUTE WEIGHT UPON THE FORM OF THE ELECTROCARDIOGRAM; FROM A THEORETICAL STANDPOINT.

Typical preponderance curves bear a striking resemblance to the curves of bundle branch block; in the one instance the effects of one ventricle overbalance the effects of the other; in the other instance the effects of one ventricle precede the effects of the other. When these curves are analyzed it appears that their peculiarities of form are due chiefly to the abnormal position of the major electrical axis. Now the position of the major electrical axis is dependent upon the relative magnitude, and not upon the difference in magnitude, of the potential differences produced by the two ventricles. Other factors being the same, the form of the electrocardiogram (i. e. the direction of the major electrical axis) should vary with the L-R ratio; the amplitude of the electrocardiogram should vary with the weight of the ventricular muscle. Why, then, do we find that large hearts

are more prone to yield preponderance curves than are small hearts with equally abnormal L-R ratios? In the first place, it is probable that other factors, such as the arrangement of the ventricular conducting system, which influence the form of the electrocardiogram, disturb the effect of the ratio L-R less when the heart is greatly hypertrophied than when it is of relatively normal size. In the second place, although the normal right ventricle produces effects of greater amplitude, in proportion to its mass than the left, it does so, according to our theory, because of certain factors previously mentioned which can hardly escape modification when the heart hypertrophies.

COMPARISON OF THE L-1.8 R VALUE AND THE ELECTROCARDIOGRAM.

Since it is usual, in judging the degree of preponderance shown by the electrocardiogram, to consider the amplitude as well as the direction of the chief deflections and since preponderance curves are more prone to occur with large than with small hearts, it seems logical, when comparing electrocardiographic and postmortem observations, to substitute for the ratio L-R some figure which is in part dependent upon the ventricular weight. Such a figure may be obtained by subtracting, from the weight of the left ventricle, 1.8 times the weight of the right.

In Table V we have arranged our cases in the order determined by the L-1.8 R value, and in Table VI we have arranged in the same way the nine cases published by Lewis, the six cases published by Cotton, and all our own cases (9) in which the ventricular weight exceeds 250 grams. These tables indicate that, on the whole, there is a closer relation between the form of the electrocardiogram and the L-1.8 R value than between the form of the electrocardiogram and the ratio L-R. We find, for instance, definite or questionable preponderance in all but one of the seven cases (Table V) in which the L-1.8 R value is greater than 70 or less than -70, and the discordances which occur in cases with abnormal L-R ratios but relative normal ventricular weights are rendered much less conspicuous. Nevertheless, the L-1.8 R value and the form of the electrocardiogram fail to correspond in many instances and occasionally notable discrepancies between them are encountered. Some of these are due, perhaps, to the failure of the normal 1-1.8 ratio to hold when the heart hypertrophies; others are probably due to factors still to be considered.

ERRORS IN DETERMINING THE RATIO L-R. In a previous section of this article, we have estimated the maximal error in the ratio L-R due to inaccuracy in sectioning the heart to be about 0.18. A case which falls near the upper limit of a group (page) may, had the cuts been made without error, have fallen in the next higher group; a case

which falls near the lower limit of a group may have fallen in the next lower group. It is clear, therefore, that, even if we assume that the maximal error occurred in every case, our chief conclusions as to the relation between the form of the electrocardiogram and the ratio L-R would not be greatly altered. But as shown by Table I and by a comparison of our normal averages with those of Lewis (Table IV) the error in determining the L-R ratio is usually very small.

CHANGES IN THE RATIO L-R BETWEEN THE LAST ELECTROCARDIOGRAPHIC EXAMINATION AND DEATH.

It has been shown by Stewart (14) that when aortic insufficiency is produced in dogs, the heart may be definitely hypertrophied at the end of the first week; comparatively little hypertrophy takes place after the fourth week. Unless, therefore, the final electrocardiogram is taken within a few days of death, a change in the ratio L-R may take place after it is made.

In the majority of our cases only a few days elapsed between the electrocardiographic examination and death; furthermore, the disease process present was in most instances of long standing so that it is improbable that hypertrophy of the heart was taking place rapidly when our observations were made. In those cases in which the patient was under observation for a long time and in which a series of curves was made, all of the curves were usually almost identical in form and in no instance did they indicate a progressive increase or decrease in right or left ventricular preponderance.

VARIATIONS IN THE POSITION OF THE HEART. It has been held by some that those abnormalities of the electrocardiogram which are now believed to be due to preponderant hypertrophy of the one or the other ventricle might arise through abnormal variations in the position of the heart. That somewhat similar curves may be produced by rotation of the heart about its anterior-posterior axis may be demonstrated in a very simple way. (Method of Dr. F. N. Wilson, personal communication). To determine the effect of counterclockwise rotation of 60 degrees it is necessary only to substitute lead II for lead I, lead III for lead II, and lead I, inverted, for lead III; the inverted lead must be read from right to left. By repeating this process the effect of rotating the heart 120, 180, or 240 degrees may be seen. By reversing the process the effect of clockwise rotation may be estimated. If these procedures are carried out upon a normal electrocardiogram, it will be seen that counterclockwise rotation of the heart gives a picture resembling that of left, and clockwise rotation, a picture resembling that of right ventricular preponderance. It will be evident, however, that rotation of the heart produces abnormalities of the P and T waves of the electrocardiogram which do not occur in preponderance

curves and that, furthermore, in order to produce curves which resemble many of the curves obtained from patients with preponderant hypertrophy of one ventricle, a much greater rotation of the heart is necessary than could possibly take place in the body. This has already been pointed out by other writers (5 and 9). It is clear, then, that rotation of the heart about an anterior-posterior axis can not account for preponderance curves of typical form. It is also known that displacement of the heart without rotation, such as is brought about by pleural effusion, or pneumothorax, has but little effect upon the form of the electrocardiogram. Regarding the effects of rotation of the heart about its long axis or about a vertical axis, we know very little.*

That the form of the normal electrocardiogram is to a considerable degree dependent upon the position of the heart is well known. It is a matter of common observation that short stout individuals (hypersthenic habitus) with transversely placed hearts usually yield electrocardiograms in which lead III is the lead of least amplitude, not only with respect to the QRS group of the electrocardiogram, but also with respect to P and T. It is in this type of individual that QRS of lead III is often of bizarre form; the inversion of T3 in association with these peculiar QRS complexes, already known (10), may be attributed to the transverse position of the heart. Individuals of slender build with vertically placed hearts (asthenic habitus) usually yield electrocardiograms of the opposite type in which lead I is the lead of least amplitude. In both types of individuals notable exceptions occur.

The effects of abnormal variations in the position of the normal heart upon the form of the electrocardiogram may be estimated by studying the effects of deep inspiration and forced expiration. Thirty experiments of this type were carried out by Dr. F. N. Wilson (personal communication) on a series of healthy male subjects. To give some idea of the range of the effects observed, the results of five of these experiments are given in Table VII. From these figures, it is clear that positive indices may become negative as the result of deep inspiration and that negative indices may become positive as the result of forced expiration. Normal curves in which lead III is the lead of least amplitude are almost always converted by deep inspiration into curves in which lead I is the lead of least amplitude. Sometimes, though less often, curves of the latter type are converted into curves of the former type by forced expiration. In our experience, normal curves have never been converted by respiratory experiments into curves in which lead II was the lead of least amplitude. In some instances normal curves are converted by forced expiration into curves which suggest questionable or slight left ventricular preponderance (cases 551 and 527 Table VII). Deep

(Note):—Boden and Neukirch (Pflüger's Archiv, 1918, clxxi, 146) believe that preponderance curves may be produced by rotation of the heart about its long axis. Their experiments upon the isolated human and animal heart are not, it seems to us, convincing.

inspiration, on the other hand, almost never converts a curve of the normal type into a curve suggestive of right ventricular preponderance; this is because inspiration almost invariably reduces the amplitude of all the deflections of lead I, even when this effect would not be anticipated. Thus in case 630 (Table VII) SI is smaller in inspiration and larger in expiration although the principles of the equilateral triangle would lead us to expect the opposite in each instance. This is probably because the respiratory changes in the position of the heart consist in rotation about its long axis as well as about an anterior-posterior axis.

Of the effect of respiratory changes in the position of the heart upon the form of the abnormal electrocardiogram we have made no systematic study; the opinions expressed below are based on many isolated observations but further experience may require us to revise them. Typical right preponderance curves are seldom very much altered by respiratory experiments; contrary to what might be expected, inspiration often renders the signs of right preponderance less, rather than more, conspicuous. Left ventricular preponderance curves, on the other hand, are often changed profoundly. Curves in which lead I is the lead of least amplitude (with reference to QRS) are frequently converted into curves of the normal type by deep inspiration. Curves in which lead II is the lead of least amplitude are often converted into curves in which lead III is the lead of least amplitude, but rarely into curves of the normal type (i. e., curves in which R is tallest in lead II). Some of the more conspicuous changes in the form of the abnormal electrocardiogram, produced by forced respiration, that we have encountered are shown in Figs. 5 and 6. In Fig. 5 are shown curves indicative of questionable left ventricular preponderance which was converted by deep inspiration into a curve indicating a tendency toward right ventricular preponderance. In Fig. 6 is shown a striking example of the effect of change of the heart's position by respiratory experiments. In this instance the direction of QRS of lead III of a curve, indicating definite left ventricular preponderance (index 36), was reversed by deep inspiration. It is evident, then, that curves indicative of definite left ventricular preponderance, more especially those in which the major electrical axis is nearly horizontal, may be converted into curves of the normal type by a change in the position of the heart such as takes place when a deep breath is taken.

TO SUM UP:—Variations in the position of the normal heart account for many, though not all, of the usual variations in the form of the normal electrocardiogram from one individual to another.

Abnormal or unusual variations in the position of the normal heart may give rise to curves indicative of questionable left ventricular preponderance; these curves are usually within or just without the normal range and are of the type

in which lead III is the lead of least amplitude. Variations in the position of the normal heart seldom give rise to curves indicative of right ventricular preponderance.

The signs of left ventricular preponderance may be absent, in cases in which they would ordinarily be present, when the heart is unusually pendulous. These signs may be exaggerated when the heart is transversely placed. The signs of right ventricular preponderance appear to be less often disturbed by such changes in the position of the heart as are dependent upon changes in the level of the diaphragm than are the signs of left preponderance.

DIFFERENCES BETWEEN THE EFFECTS OF PREPONDERANCE OF THE LEVOCARDIOGRAM OR DEXTROCARDIOGRAM AND THE EFFECTS OF ROTATION OF THE HEART. The effects of rotation of the heart may often be distinguished from those of preponderance of the levocardiogram or dextrocardiogram; the former differ from the latter in several respects.

1. As has already been noted, rotation of the heart produces changes in P and T similar to those which it produces in QRS; preponderance of the levocardiogram or dextrocardiogram does not effect P; its effect upon T is uncertain but in any case it does not affect T in the same way as does rotation of the heart. In the application of this knowledge, however, considerable difficulty arises. Hearts which yield preponderance curves are prone to show other electrocardiographic abnormalities due to myocardial changes in both the auricular and ventricular muscle. Patients with heart disease often receive digitalis which produces deformities of T. Very large hearts are usually transversely placed so that a combination of the effects of rotation and preponderance is obtained. Little dependence can be placed upon the form of P and T, therefore, when the heart is abnormal, in judging the position of the heart and its effects upon the electrocardiogram.

2. It has also been pointed out that preponderance of the levocardiogram or dextrocardiogram may produce changes in QRS which could not be simulated by such an amount of rotation of the heart as is possible.

3. Unlike ventricular preponderance, displacement of the heart can not increase the magnitude of the greatest manifest potential difference produced by the heart. Since the maximal deflection represents not less than 85% (the cosine of 30 degrees) of the greatest manifest potential difference, rotation of the heart about an anterior-posterior axis can not materially increase the amplitude of the tallest deflection of the three leads.

4. It has been shown (8) that preponderance curves show on analysis a fairly uniform rotation of the electrical axis; when the dextrocardiogram preponderates, the rotation is clockwise; when the levocardiogram preponderates, it is

counterclockwise. Normal curves show some movement of the electrical axis but, usually, of a much less uniform kind.

In so far as ventricular preponderance produces characteristic rotation of the electrical axis, its effects may be distinguished, from those of rotation of the heart about an anterior-posterior axis, which does not affect the movements of the electrical axis, by analysis. For ordinary use this is a laborious and impractical methods. Curves which show rotation of the electrical axis, during the period when the manifest potential difference is near its maximum, may, however, often be distinguished from those that do not by inspection. Certain simple tests may also be applied. The failure of the electrical axis to rotate during the inscription of R causes the chief deflections of the three leads to be in phase; they will therefore obey the rule that

Lead I plus Lead III equals Lead II.*

In preponderance curves, on the other hand, corresponding peaks are seldom in phase; if the rotation of the electrical axis is clockwise, the greatest deflection of lead III precedes that of lead I; if the rotation is counterclockwise, this order is reversed.

Curves that show uniform rotation of the electrical axis do not, as a rule, show notching of the QRS of least amplitude; such notches are due to irregularities in the movement of the electrical axis and especially to changes in the direction of its rotation (16). For obvious reasons these are prone to occur in curves of the normal type. When not present, in the original curves, notches may sometimes be brought out by a respiratory test. The respiratory test shown in Fig. 6 gave rise to a notch due to a change in the direction of rotation of the electrical axis from counterclockwise to clockwise; this curve is not a typical preponderance curve in so far as it does not show uniform rotation of the electrical axis.

THE EFFECTS OF THE POSITION OF THE HEART UPON THE ELECTROCARDIOGRAM IN OUR SERIES OF CASES. With the considerations outlined above in mind, we have gone over our cases to determine if possible the part played by variations in the position of the heart causing the observed discordances between the ratio L-R and the form of the electrocardiogram. In Table V we have tabulated such data as can be given in condensed form. Ascites, aneurysm, hydrothorax, and other factors which may cause displacement of the heart are noted. In each case the difference between the sum of the chief deflections of leads I and III and the chief deflection of lead II is given; it may be taken as a rough measure of the lack of synchronism between the chief peak, and

*(Note).—An examination of 30 normal curves by Dr. F. N. Wilson (personal communication) from this standpoint gave the following results:—

R1 plus R3 equalled R2, 5 instances

R1 plus R3 equalled R2 plus or minus 0.1 millivolt, 16 instances.

R1 plus R3 equalled R2 plus or minus 0.15 millivolt, 1 instance.

R1 plus R3 equalled R2 plus or minus 0.25 millivolt, 6 instances.

R1 plus R3 equalled R2 plus or minus 0.35 to 0.75 millivolt, 2 instances.

consequently of the amount of rotation of the electrical axis during the time when the manifest potential difference was near its maximum.* In about one-half of our cases X-ray plates were available; in each of these instances we have measured the angle between the long diameter of the ventricular shadow and the horizontal, the interval which elapsed between the X-ray examination and death is given so that the time of the X-ray examination with reference to the time of the electrocardiographic examination is shown. It should be remembered that the X-ray plates were made with the patient in the erect posture, while electrocardiographic curves were made with the patient in the supine or in the sitting posture. Angles measured on teleroentgenograms are starred.

It would not be profitable to enter into a discussion of the part played by the position of the heart in determining the form of the electrocardiogram in individual cases; in a great many instances no definite opinion can be formed. It may be responsible for many of the minor discordances between the ratio L-R and the form of the electrocardiogram that we have encountered (See cases 29 and 55); and it may have exaggerated some of the major discordances. It does not offer a satisfactory explanation of such discordances as occur in cases 2, 3, and 42. In our opinion it is to be considered but one of many factors which disturb the relation between the form of the QRS group and the relative weight of the two ventricles.

DISTURBANCES OF INTRAVENTRICULAR CONDUCTIVITY AND THE ARRANGEMENT OF THE VENTRICULAR CONDUCTING SYSTEM. The aberrant electrocardiograms produced by lesions which prevent the passage of the impulse through one of the chief branches of the His-bundle and their superficial resemblance to preponderance curves require no comment. Recently, we have shown that lesions which delay the passage of the impulse through one of the bundle branches may produce curves which resemble preponderance curves very closely (16). This had previously been suspected by others (7 and 11). Fahr advanced the theory that all preponderance curves were produced in this way. He believes that the curves which are now generally attributed to preponderance of the levocardiogram are in reality due to preponderance of the dextrocardiogram produced by dilatation of the left ventricle and consequent prolongation of the left bundle branch and its arborizations, delaying the passage of the impulse through the left ventricular conducting system. He claims to have made certain measurements of the relative length of the right and left conducting paths, which support his hypothesis. Rothberger and Winterberg (13) have shown that section of the anterior subdivision of the left bundle-branch in the dog produces curves not unlike

* (Note) :—In studying our cases with reference to the amount of rotation of the electrical axis shown by the curves, we have not considered this figure by itself; it has been used in connection with inspection of the electrocardiograms.

curves of left ventricular preponderance in man; section of the posterior subdivision of this branch produces curves of more or less opposite type.

It cannot be denied, therefore, that curves similar to those of preponderance may be produced by lesions which interfere with the normal spread of the impulse through the ventricular conducting system. But that all preponderance curves arise in this way cannot be held at present for the following reasons:—

1. The preponderance curves of the new-born infant cannot be explained on this basis. It may be that the ventricular conducting system of the new-born infant differs from that of the adult; at present, however, we have no foundation for such an assumption.

2. The curves which result from disturbances of intra-ventricular conductivity are almost invariably diphasic when they differ greatly from curves of the normal type. This is more especially the case when QRS is of great amplitude; in leads of very small amplitude QRS and T may have the same direction even in complete bundle branch block. Although preponderance curves show a greater tendency to be diphasic than do normal curves, they are often not diphasic, even when of the most exaggerated type (Fig. 1 A).

3. Although conduction disturbances often give rise to curves of unusually large amplitude, they seldom, if ever, give rise to curves of enormous amplitude such as are sometimes seen in pulmonary stenosis (Fig. 1 A).

4. Preponderance curves seldom or never develop suddenly. Bundle branch block curves, although they are much less common than preponderance curves, have frequently been observed to develop over night; so far as we know, preponderance curves have never been observed to develop within a brief period of time. They are much more stable than we should expect, if they were due to lesions of the conducting system. Even if they are to be attributed to dilatation of the one or the other ventricle as Fahr believes, we should expect them to appear or disappear quickly in some cases.

5. The relation of right ventricular preponderance curves to pulmonary stenosis and mitral stenosis, and of left ventricular preponderance curves to aortic insufficiency and to chronic hypertension, is difficult to explain, if we attribute preponderance curves to disturbances of intraventricular conductivity, unless Fahr's hypothesis be adopted.*

With the hope of gaining some further evidence bearing upon the influence of relative dilatation of the two ventricles

*(Note):—This is not the place to discuss Fahr's general hypothesis, that what we now regard as left ventricular effects are in reality right ventricular effects, in detail. It is based almost wholly upon theoretical consideration which we regard as precarious. Fahr has disregarded many facts that are not in accord with this theory; the form of the bundle branch block curves of the Rhesus monkey (8) for example. Until he is able to bring his theory more into accord with past observation and until he is able to support it by new experimental evidence, we shall find difficulty in accepting it.

upon the form of the electrocardiogram, we measured the capacity of each ventricle in a number of our cases by a method which has already been described. These measurements are given in Table V. It will be seen at once that the relative dilatation of the two chambers, as determined by this method, plays no appreciable part in determining the form of the ventricular complex. It is unquestionably true, however, that the volumes of the ventricular cavities after death bear no definite or constant relation to their volumes during life when the electrocardiograms were made. We include the measurements, realizing that they are of doubtful value, because such measurements have been suggested and because they have a bearing upon the type of measurements that Fahr claims to have made. We include, also, measurement of the thickness of the lateral wall of each ventricle. It will be seen that between these and the measurements of the ventricular capacities there is a sort of reciprocal relation; dilated ventricles have thin walls; contracted ventricles, thick walls.

That lesions of the ventricular conducting system were responsible for lack of agreement between the ratio L-R and the form of the electrocardiogram in some of our cases is probable; in none of our cases can we feel certain that this was the case. None of the electrocardiograms show definite prolongation of the QRS interval; only two show an increase in the P-R interval and in both of these it is very slight (cases 8 and 25). In two instances the electrocardiograms are definitely diphasic (cases 1 and 42) and conduction defects may have

ARCHITECTURE OF PURKINJE NETWORK

There is a factor related to disturbances of intraventricular conduction that requires discussion here; we refer to the arrangement of the ventricular conducting system. It has been shown (8) that in the dog striking variations in the architecture of the conduction system of the left ventricle occur. That similar variations in the arrangement of the ventricular conducting system occur in man is hardly to be doubted; it is probably that it is upon the architecture of this system that the individuality of the normal electrocardiogram chiefly depends. It seems likely that in most individuals both ventricles do not receive the impulse at exactly the same time; in those in which Q is largest in lead III and S in lead I, the right ventricle may receive the impulse in advance of the left; in other cases the left ventricle precedes the right. In those individuals who normally exhibit curves of the preponderance type, the precedence of one ventricle over the other may be unusually great, although not of the same grade as that seen in pathological cases.

We have already pointed out that the position of the heart has an important influence upon the form of the normal electrocardiogram; its effect is usually combined with that of the arrangement of the ventricular conducting system in such

a way that the most important of the two factors can not be determined. Occasionally, however, this is possible. In Fig. 7 is shown the electrocardiogram of a young woman of unusually stout build; in this instance the form of the electrocardiogram is determined largely by the transverse position of the heart. P, QRS, and T are all of small amplitude in lead III; QRS and T are inverted. In Fig. 8 the electrocardiogram of a young woman of average build is shown; in this instance the form of the electrocardiogram, because of its diphasic character, is determined mainly by precedence of the right ventricle. This conclusion is based on the results of a previous experimental study (17) T3 is inverted here also, but in this instance R3 is tall; T is largest in lead I which shows a conspicuous S deflection; Q is present in lead III alone. The form of P indicates that the heart is of the pendulous type, but the position of the heart cannot account for the diphasic character of leads I and III.

If it be admitted that the arrangement of the ventricular conducting system may be such that one ventricle receives the impulse slightly in advance of the other; then it seems probable that certain individuals develop preponderance curves as a result of alteration of the ratio L-R more readily than others. If, for instance, some peculiarity of the conducting system gives rise in a certain individual to curves which suggest slight right ventricular preponderance, it will probably take a much greater relative preponderance of the left ventricular muscle to produce the signs of left ventricular preponderance, than in an individual with a conducting system of the opposite type. Our knowledge is insufficient, however, to determine how these two factors, the arrangement of the conduction system and the relative weight of the two ventricles, will react with one another under various conditions.

REMARKS UPON INDICES

During the last few years, a belief that there is a close relation between the form of the electrocardiogram and the ratio L-R has lead several observers to advocate the use of numerical indices to express the degree of right or left ventricular preponderance shown by the electrocardiogram. White and Bock (15) advocate an index similar to the one used in this article. Carter and Greene (1) prefer an index based upon Einthoven's equilateral triangle; their index is intended to express in degrees the position of the major electrical axis; whether it does so or not need not be discussed here. We have computed this index in each of our cases; it is given in Table V. None of the indices so far proposed are of any precise value in distinguishing between the effects of rotation of the heart and of conduction defects, on the one hand, and the effects of an abnormal L-R ratio, on the other. They are greatly inferior to simple inspection of the electrocardiogram.

MICROSCOPICAL PATHOLOGY IN CONTRAST TO THE ELECTROCARDIOGRAPHIC FINDINGS.

There has been a tendency to interpret clinically slight abnormalities in the electrocardiographic curves in the terms of anatomical lesions in the myocardium. The bases for these ideas have not infrequently been quite unsound because of the uncontrolled and limited pathological data. Anatomical findings are usually recorded in only a very small percentage of cases, and there is usually agreement with the hypotheses in only about half of these. (16) The cases with similar electrocardiographic findings and the absence of the anatomical lesions are often only lightly considered as exceptions and disregarded. Likewise, cases with similar anatomical lesions and the absence of the electrocardiographic findings are often not seriously enough sought out and considered.

The possible physiological cause for the electrocardiographic change is sometimes lost sight of, as, for instance, in the case of the notching of the QRS, the mechanism of the physiologic production of which was established by Wilson (17). The fallacy of attributing certain electrocardiographic findings to arborization block with a subendocardial fibrous myocarditis was also pointed out by Wilson (17).

The association of small complexes with myocardial lesions is not constant or reciprocal as a study of Tables II and III definitely shows.

The change in the direction of the T wave, especially in leads I and II, to negativity, producing at times definitely diphasic curves is suggestive, but as yet not established, evidence of myocarditis. Digitalis, which is universally administered in heart cases, produces inversion of the T wave, and consequently almost always complicates the interpretation of the findings.

In our cases 1 and 47, in which the curves were definitely diphasic, suggesting possible lesions in the branch of the His bundle, there were conspicuous microscopical findings of myocardial disease.

Prolongation of the P-R interval, indicating disturbed conduction in the His bundle, is fairly reliable evidence of heart disease. A very localized lesion is the uncommon and, a wide spread lesion the usual accompaniment of defective auriculoventricular conduction.

In our cases 9 and 25, in which there was an abnormal prolongation of the P-R interval, there were microscopical findings of advanced myocardial disease.

Auricular flutter and fibrillation are found practically only in cases of severe heart disease, as our cases 8, 50 and 58 seem to indicate or substantiate.

Thus, conduction disturbances in either the auriculoventricular or intraventricular specialized tissue, and severe disturb-

ances in rhythm, such as auricular fibrillation and flutter, are associated, as a rule, with definite myocardial disease. On the other hand, the converse does not hold, in that, a study of the last column of Table II and Table III shows case after case with microscopical evidence of myocardial disease and a study of the electrocardiographic curves reveals no accompanying abnormalities.

CONCLUSIONS

Summary

The relative weight of the two ventricles is but one of many factors which influence the form of the ventricular complex of the electrocardiogram. Its influence predominates only when the heart is greatly hypertrophied. There is no definite relation between the form of the ventricular complex and the relative weight of the two ventricles when the ventricular weight is below 250 grams.

The chief factors which disturb the relation between the been present in these.

form of the electrocardiogram and the relative weight of the two ventricles, so it is suggested, are; (1) variations in the position of the heart, (2) variations in the arrangement of the ventricular conducting system, and (3) disturbances of intraventricular conduction.

The form of the normal electrocardiogram is not determined by the relative weight of the two ventricles; it is chiefly dependent upon the position of the heart and upon the arrangement of the ventricular conducting system; sometimes one, sometimes the other of these factors exerts the greater influence.

Electrocardiographic abnormalities cannot be interpreted in the terms of anatomical lesions. A normal electrocardiogram does not necessarily mean normal heart muscle. Conduction disturbances and severe arrhythmias are usually found only in hearts in which there is extensive myocarditis. The converse, however, is not true, in that there are many cases of severe myocarditis without electrocardiographic abnormalities.

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TABLE I.—Comparison of methods.

Serial and Case No.	METHOD A (Disc.)					METHOD B.				LEWIS' METHOD.					Differences.	
	Vent. Wt. Gms.	R Vent. Gms.	L Vent. Gms.	S Sep- tum Gms.	L/R	Vent. Wt. Gms.	R Vent. Gms.	L Vent. Gms.	L/R	Vent. Wt. Gms.	R Vent. Gms.	L Vent. Gms.	S Sep- tum Gms.	L/R		
2 22	185					182	44 0	138 0	3 14	177	31 9	114 6			0 03	0 45
4 48	379	37 0	119 3	29 0	3 22	380	121 0	259 0	2 14	351	74 7	210 5	65 6	2 82	0 68	0 38
6 47	346	90 5	228 3	60 0	2 52	346	105 0	241 0	2 29	374	77 3	232 4	53 8	2 62	0 33	0 26
7 37	99	84 5	215 2	47 0	2 55	98	32 5	65 5	2 02	91	21 9	56 4	13 1	2 57	0 55	0 28
10 36	139	26 5	61 0	11 5	2 30	136	42 5	93 0	2 18	130	32 0	79 3	18 7	2 48	0 30	0 01
17 40	297	38 5	84 3	15 3	2 19	302	103 7	193 1	1 78	276	74 6	164 8	36 4	2 20	0 42	0 13
18 41	167	91 3	174 5	30 7	1 91	169	58 3	110 5	1 90	163	45 6	97 5	29 0	2 14	0 24	0 06
A-47-Z		51 0	100 0	16 1	1 90	414	138 5	275	1 94	414	115 0	242 5	60	2 11	0 17	
19 51						143	50 3	92 5	1 85	140	39 3	79 6	20 8	2 02	0 17	
20 52						184	67 5	116 4	1 73	178	50 8	101 1	26 3	1 99	0 26	
A-58-Z						63	21 5	41 5	1 93	63	17 5	24 5	11 5	1 97	0 04	
23 44	142	40 0	77 7	24 0	1 94	143	50 0	93 2	1 85	138	37 6	73 7	26 7	1 96	0 11	0 09
24 29	339	104 0	193 0	42 2	1 86	335	118 0	217 0	1 84	323	91 6	177 7	54 1	1 94	0 10	0 02
25 13	385	115 3	213 0	57 0	1 85	390	140 5	240 7	1 76	372	104 7	239 7	65 0	1 94	0 18	0 09
A-78-Z						190	71	113	1 66	185	57 5	100	23	1 93	0 27	
27 54						120	43 9	76 9	1 75	119	33 7	64 6	20 8	1 92	0 17	
28 49	197	69 0	112 7	17 3	1 64	193	75 5	121 7	1 61	195	53 1	105 5	20 6	1 83	0 22	0 03
29 57						111	42 3	68 2	1 61	107	33 1	63	13 3	1 82	0 21	
A-38-7						24	8 25	14 7	1 79	23	6 75	12 5	3 5	1 81	0 02	
30 35	160	51 7	87 0	20 8	1 68	161	61 3	103 0	1 64	152	46 0	82 7	23 3	1 80	0 16	0 04
33 46	92	32 5	51 0	8 5	1 60	89	35 7	53 3	1 49	87	27 0	47 6	12 8	1 75	0 26	0 11
A-97-Z						97 5	37 5	59 5	1 59	95	30	51	14 8	1 70	0 11	
35 21	146	48 0	79 0	18 7	1 65	145	60 0	85 0	1 42	139	43 1	73 5	22 5	1 70	0 23	0 23
38 59						169	69 8	99 2	1 42	160	49 2	83 6	26 9	1 70	0 28	
39 53						204	85 8	119 4	1 39	200	63 9	107 8	28 6	1 69	0 30	
43 42	152	53 7	80 5	18 6	1 50	155	64 0	90 6	1 42	146	47 9	75 9	20 8	1 61	0 19	0 08
44 55						216	85 6	130 2	1 53	215	71 6	115 4	27 6	1 61	0 08	
45 38	145	52 5	74 9	18 0	1 43	143	60 0	83 0	1 39	137	43 7	69 9	23 6	1 60	0 21	0 04
A-105-Z						233 5	90 5	142	1 57	233	77	121 5	31 5	1 58	0 01	
49 45	181	68 2	98 0	14 8	1 44	182	76 5	104 5	1 37	167	53 5	91 1	17 5	1 56	0 19	0 07
A-50-Z						160	67	102	1 46	165	53 5	82 2	30	1 53	0 07	
A-37-Z						138	58	80	1 38	130	47	72	11	1 53	0 15	
A-72-Z						146	60	86 5	1 44	144	48	73	23 5	1 52	0 08	
A-43-Z						86 5	36 5	50 5	1 38	82	27	41	14	1 52	0 14	
51 39	197	74 3	103 7	19 0	1 39	196	85 5	110 7	1 38	186	64 1	96 8	25 3	1 51	0 21	0 09
A-40-Z						235	104	150	1 44	250 5	85	127 5	38	1 50	0 06	
A-71-Z						226	90	137	1 52	226	78	116	31	1 49	0 035	
53 50	160	60 0	82 3	17 3	1 37	158	67 0	91 0	1 36	145	50 4	73 7	20 7	1 46	0 10	0 01
A-49-Z						105	44	60 5	1 37	104 5	36 2	52 2	16 2	1 44	0 07	
A-35-Z						71	31	41	1 32	70	25	35 5	10	1 42	0 10	
A-69-Z						143	60 5	83 5	1 38	143	52 5	74	17	1 41	0 03	
A-49-Z						45	18 5	25 5	1 38	44 2	16 0	22 5	6 25	1 38	0 00	
56 43	131	50 0	67 7	13 0	1 36	133	56 7	75 0	1 32	125	45 1	62 3	17 7	1 38	0 06	0 04
A-96-Z						205	85	115 5	1 36	196	70	95 5	30 5	1 36	0 00	
A-86-Z						272	112 5	144 5	1 23	265	100 5	126 5	38	1 26	-0 02	
A-95-Z						60	26 5	33	1 24	57 5	22	27 5	9 5	1 22	-0 02	
A-102-Z						189	89 5	103	1 15	185	76	85	27 5	1 20	0 05	
57-56						215	104 1	111 2	1 07	203	80 7	96 3	30 7	1 19	0 12	
53-2	273	117 5	127 7	28 0	1 09	276	132 7	143 7	1 03	261	107 3	120 2	33 3	1 12	0 04	0 01
A-43-Z						15	7 5	7 0	0 93	14 6	6 6	6 25	1 75	0 95	0 02	
Aver.	205.3	64.5	110.9	25.1		176.3	68	179		170	53.5	91.8	25		0.162	

TABLE II.—*Pathological findings.*

No.	Case No.	Age, Sex.	Ht. cm.	Wt. kg.	Heart wt. gms.	Vent wt. gms.	R	L	S	L/R	V
1	58	39 M	165	54	537	387	64.7	247.6	75.0	3.83	
2	22	14 F	145	30	290	177	31.9	114.6	30.0	3.59	
3	19	47 M	175	57	420	224	48.8	139.9	35.2	2.86	
4	48	35 M	167	57	520	351	74.7	210.5	65.6	2.82	
5	3	32 M	175	75	700	391	85.9	232.5	72.3	2.72	
6	47	38 M	176	62	615	334	77.3	202.4	53.8	2.62	
7	37	60 F	160	38	168	91	21.9	56.4	13.1	2.57	
8	1	78 F	135	34	540	158	36.3	92.0	29.3	2.53	
9	33	60 F	162	40	385	194	45.1	113.0	36.0	2.50	
10	36	76 M	183	43	205	130	32.0	79.3	18.7	2.48	
11	7	48 M	157	45	700	427	105.0	259.4	62.6	2.46	
12	9	44 M	185	64	305	163	39.4	96.7	26.5	2.46	
13	24	48 F	161	34	285	101	26.2	61.8	12.4	2.36	
14	6	57 M	175	48	315	181	48.2	108.7	24.6	2.25	
15	10	59 M	165	48	300	146	37.8	85.0	23.0	2.25	
16	31	75 F	145	30	285	128	34.0	74.7	19.3	2.20	
17	40	37 F	150	55	480	276	74.5	164.8	36.4	2.20	
18	41	49 M	160	58	350	163	45.6	97.5	20.0	2.14	

TABLE II.—*Pathological findings.*

Kidney Lesions.	B. P. Systolic.	B. P. Diastolic.	General Remarks.	Valvular Defects.	Myocardial Lesions.
0 gms. L. 215 gms. Surf. sl. nodular. Chr. dif. nephritis.	240 280	160 170	Alb. Retinitis. Chr. Uremia. Mod. Sclerosis Coronaries, Aorta, etc.	None.	Cloudy swelling, fatty deg. infiltration.
3 gms. L. 52 gms. Small gran. kidneys. Chr. dif. nephritis.	275	190	Alb. Retinitis. Right Empyema. Some scl. of aorta. Aur. mural thrombi, ball thrombus in aorta.	None.	Simple necrosis. Cl. swelling, fatty deg. inf. CT increase.
0 gms. L. 210 gms. Ac. and chr. nephritis.	132	58	Ac. and chr. pericard. Mural thrombus. Aortitis. Early aneurysm (Strep. viridans).	Aortic and mitral vegetations. Aortic regurg. (Strep. viridans.)	Chr. myocarditis. Cl. swelling. Fatty deg. infl. CT prolif. Simple necrosis.
0 gms. L. 80 gms. Small red kidneys. Chr. dif. nephritis.	228	150	Alb. retinitis. Uremia. Great coronary and periph. sclerosis. Aorta free.	None.	Sclerosis of vessels. Mononuc. plasma cell inf. esp. epicardial?
diff. deg. parench. nephritis. Large white kidney.	185	140	Alb. retinitis. Uremia. Anasarca. Fibrino-pur. pericarditis and pleurisy.	None.	Fib-inc-pur. epicard. Cl. sw. Simple and coag. necrosis. Chr. myocarditis.
0 gms. L. 140 gms. Art. scl. in kidn. Chr. dif. nephritis.	265	145	Alb. retinitis. Mod. gen. art. scl. Mod. aortic sclerosis.	Small well organized aortic vegetations, no regurgitation.	Slight atrophy. Cloudy swelling.
0 gms. L. 110 gms. Scl. arteria. Glom. fibrotic. Sl. interstitial increase. Chr. dif. nephritis with cysts.	118	76	Ascites. Considerable periph. and aortic scl.	None.	Brown atrophy and widespread fibrosis.
5 gms. L. 155 gms. Lt. hydro-nephrosis. Chr. dif. nephritis.	170	110	Aur. flutter and fibril. Heart considerably displaced. Gen. art. scl. Syph. aortitis with large aneurysm.	None.	Chr. endo. and subendo cardial fibrosis. Cl. sw. smp. necros. Scl. and fibrosis.
0 gms. L. 255 gms. Rt. pyonecrosis. Lt. hypertrophy.	180	100	Mod. scl. of aorta and coronaries.	Fresh aortic vegetations. No regurg.	Chr. endo. and myocard. Sub-endocardial fib. Cl. sw. Poly inf. bacterial colonies.
and L. 275 gms. (comb.). Art. of vessels. Fibrosis of many m.	140	70	Ascites. Emphysema. Mod. atheroma and some calcif. of aorta.	Mitral and aortic thickenings. Mitral regurg.	Thickening of endocard. C. T. inf. calcif. mono. inf. bacterial colonies.
0 gms. L. 120 gms. Art. scl. nephritis.	180	75	Transient aur.fib.cardiac failure. Aorta atheromatous.	Ac. and chr. veg. aortic, mitral and triusp. endocarditis (S. Viridans). Aortic regurgitation.	Marked cloudy swell. Mononuc. and CT infl. Endocarditis. (Sub-acute.)
5 gms. L. 180 gms. Congestion and acute deg. changes.	160	70	Base of aorta slightly sclerotic. Thromb. of rt. pulm. art. Hemorr. into vent. septum.	Patent foramen ovale. Slight fenestration of pulmonary segments.	Hypertrophy of muscle fibers. Suppurative focus at base of lt. ventricle.
and L. 940 gms. comb. Capsule thin. Surface granular. Interstitial increase. Chr. diffuse nephritis.	110	70	Pernicious anemia. Ascites. Hydrothorax (lt.) hydropericardium (sl.). Aorta atheromatous.	None.	Marked fatty deg. inf. Diffuse leucocytic infl. of myocardium.
30 gms. L. 260 gms. Bilateral hydroneph. Great increase in interstitial CT. Chr. nephritis.	260 140	110 75	Carc. of prostate. Chr. uremia. Adv. gen. scl. with calcif. of esp. aorta and coronaries.	None.	Diffuse CT. infl. Slight atrophy.
50 gms. L. 155 gms. Surface very gran. Capsule adherent. Many abs. Chr. nephritis.	140	70	Diabetes mellitus. Vent. extrasystoles. Gen. art. sclerosis.	None.	Marked fibrosis with some atrophy of muscle cells.
00 gms. L. 95 gms. Capsule thin. Surface sl. gran. Vessels scl. Interstitial tissue. Chr. nephritis.	200 130	75 40	Endarteritis obliterans (fert). Great scl. of aorta and coronaries. Lt. coronary narrow (very).	Aortic and mitral valve fibrosis. Calc. nodule in one aortic segment.	Advanced scl. of art. increase in CT.
0 gms. L. 150 gms. Scl. of small arterioles. Congestion. Ac. deg. nodular nephritis.	126	65	Infarction of lungs. Pleural effusion. Mural thrombi in lt. vent. sl.	Slight mitral and triusp. regurg. by hydrostatic test.	Many areas of dense leucocytic infl. CT increase. Thickened endocardium.
00 gms. L. 190 gms. Slight atheroma of vessels. Fatty deg.	125	90	Carc. of liver. Hemochromatosis. Ascites. Atheromatous aorta.	Slight atheroma of aortic cusp.	Brown atrophy. Marked increase in fibrous connective tissue.

TABLE II.—*Pathological findings (continued).*

No.	Case No.	Age, Sex.	Ht. cm.	Wt. kg.	Heart wt. gms.	Vent. wt. gms.	R	L	S	L/R	V P
19	51	58 M	162	45	360	140	39.3	79.6	20.8	2.02	
20	52	68 M	178	72	408	178	50.8	101.1	26.3	1.99	
21	26	17 F	146	40	235	157	44.3	87.0	25.4	1.97	
22	13	70 F	157	49	340	160	44.0	86.3	30.0	1.96	
23	44	41 F	180	79	280	138	37.6	73.7	26.7	1.96	
24	29	71 M	161	66	600	323	91.6	177.7	54.1	1.94	
25	18	63 M	157	71	700	372	104.7	202.7	65.0	1.94	
26	8	17 M	175	36	210	119	32.7	63.2	22.7	1.93	
27	54	56 F	150	35	225	119	33.7	64.6	20.8	1.92	
28	49	33 F	158	48	320	185	58.1	106.5	20.6	1.83	
29	57	26 F	160	35	187	107	33.1	60.3	13.3	1.82	
30	35	33 F	159	33	280	152	46.0	82.7	23.3	1.80	
31	11	63 M	161	37	250	111	33.0	59.5	18.7	1.80	
32	30	34 M	178	53	305	173	53.8	95.3	24.0	1.78	
33	46	54 F	165	50	205	87	27.0	47.6	12.8	1.75	
34	34	38 M	157	43	225	159	49.5	85.0	24.0	1.71	
35	21	16 M	153	37	230	139	43.1	73.5	22.5	1.70	
36	25	46 M	147	51	305	147	46.9	80.0	20.0	1.70	
37	32	31 M	175	52	400	193	60.7	103.5	29.1	1.70	
38	59	19 M	170	40	261	160	49.2	83.6	26.9	1.70	
39	53	22 M	174	61	279	200	63.9	107.8	28.6	1.69	

TABLE II.—*Pathological findings (continued).*

Kidney Lesions.	B. P. Sys- tolic.	B. P. Dia- stolic.	General Remarks.	Valvular Defects.	Myocardial Lesions.
and L. normal size. Fibrosis of any glom. Inc. in interstitial tissue. Chr. diffuse nephritis.	110	78	Carc. of esophagus. Mod. gen. art. scl. Aorta slightly scl.	None.	Slight CT increase. Much thickened endocardium with leucocyte infiltration.
10 gms. L. 120 gms. Surf. gran. any fibrotic glom. Much CT scl. vessels. Chr. dif. nephritis.	158	68	Carc. of stomach. Adv. scl. of periph. arteries and aorta.	Aortic and mitral valve fibrosis. Calcif. of mitral.	Fatty atrophy. Metastatic adeno-carcinoma with adv. deg. of surrounding heart muscle.
40 gms. L. 110 gms. Congestion and oedema.	110	35	Cardiac failure. Hydrothorax. ascites. Sl. scl. of aorta.	Chr. aortic, mitral and tricuspid. Rheumatic endocarditis with regurgitation and acute vegetations.	Small areas of endo. and myocardial fibrosis. Cloudy swelling and simple necrosis.
70 gms. L. 190 gms. Surf. gran. vessels scl. Increased CT. Chr. dif. nephritis.	150	60	Aur. fib-til. Cardiac failure. Sero-pur. pericard. Gen. art. scl. Aorta atheromatous. Coronaries scl. and calcified.	Aortic and mitral v. fibrosis. Mitral regurgitation.	Marked fatty atrophy. Areas of calcif. and leucocytic infiltration.
and L. 420 gms. comb. Essentially negative.	90	65	General peritonitis post appendectomy.	None.	Marked fatty atrophy. Cl. sw. Leuc. infil.
50 gms. L. 165 gms. Capsule b. Surf. gran. Vessels scl. Chr. dif. nephritis.	150	100	Cardiac failure. Hydrothorax. ascites. Chr. passive congestion. Atheromatous aorta and coronaries.	None.	Marked increase CT areas of myocardial necrosis, with leuc. infil. and colonies of bacteria. Thick endocardium.
50 gms. L. 170 gms. Red gran. Chr. dif. nephritis.	200 185	140 115	Alb. retinitis. Cardiac failure. Gen. anasarca. Partial heart block. Sl. atheroma of aorta.	Calcif. about mitral and tricuspid rings.	Suppurative foci. Bacterial colonies. Sub endocardial and myocardial necrosis.
10 gms. L. 120 gms. Ac. deg. angles.	118	90	Polioencephalitis. Bronchopneumonia.	None.	Sl. cloudy swelling.
and L. 220 gms. c. mb. Passive congestion only.	140	104	Mod. periph., aortic and coronary scl.	None.	Fatty atrophy. Considerable fibrosis. Dif. leuc. inf.
and L. 300 gms. comb. Congestion and cloudy swelling.	145 130	110 100	Post-partum infarction of lungs.	None.	Mononuc. inf. of myocardium. Fibroblastic proliferation.
and L. 220 gms. comb. Cloudy swelling. Simple necrosis. Fatty g.	100	65	Carc. of rt. lung. Bronchopneumonia (lt). Aorta normal.	Sl. thickening of septal mitral valve flaps. Sl. stenosis.	Marked cl. swelling. Dif. esp. subendocard. mononuc. infiltration.
90 gms. L. 220 gms. Suppurative nephritis with multiple abscesses ur. glom. nephritis.	148	70	Uremia. Fibrous pericarditis. aorta normal.	Intervent. septum defect. 6mm in diam. Patent foramen ovale.	Cloudy sw. Atrophy mononuc. inf. chr. myocarditis.
95 gms. L. 110 gms. Vessels scl. some scarring. Chr. nephritis.	150	90	Carc. of esophagus and rectum. Gen. art. scl. Dilated atheromatous aorta. Scl. coronaries.	Sl. thickenings of the mitral flaps.	Brown atrophy. Cl. sw. smp. necrosis. Thickened endocard. BV. cong. Fibroblastic prolif.
40 gms. L. 140 gms. Cloudy swelling.	115	65	Otitis media. Brain abscess. Lt. empyema. Sl. atheroma of aorta.	Sl. fenestration of aortic and pulm. cusps.	Subepicard. fatty atr. Sl. CT inc. groups of polys and monos.
60 gms. L. 150 gms. Ac. deg. angles.	115	75	Carc. of breast. Aorta normal.	None.	Abscesses in myocard. Cl. sw. endocarditis.
30 gms. L. 130 gms. Cloudy swelling.	118	94	Carc. of stomach. Ascites aorta normal.	None.	Marked cl. sw. Subepicardial. inflam. CT inc.
20 gms. L. 120 gms. Cong. cl. swelling.	100	86	Cerebrospinal meningitis, bronchopneumonia.	None.	Dif. mononuc. inf. Very much thickened endoe.
0 gms. L. 210 gms. Hyalin chg. arteriol.	145	90	Cirrhosis of liver. Syph. aortitis c large lnnom. aneurysm.	Sl. thick. of mitral and aortic cusps.	Fibrosis. Sl. plasma cell inf. Simple nec. congestion.
and L. 550 gms. comb. Fatty degeneration.	100	50	Pernicious anemia. Scl. of aorta. Fatty deg. myo.	Relat. mitral and tricuspid. regurg.	Fatty degen. of myoc. (adv). Dif. CT inf.
and L. 230 gms. comb. Cl. sw. up. nec. Mil. Tbc.	105	70	Miliary tbc. tubercles along coronaries.	None.	Miliary tub. in myoc. Cl. sw. inc. CT. Scleros.
and L. 430 gms. comb. Essentially g.	130	80	Osteomyelitis of jaw. Rt. empyema. Sl. scl. aorta.	Sl. thick. of mitral segments.	Dif. mononuc. subendo. infiltration.

TABLE II.—*Pathological findings (concluded).*

No.	Case No.	Age, Sex.	Ht. cm.	Wt. kg.	Heart wt. gms.	Vent. wt. gms.	R	L	S	L/R	F Ven Bo
40	4	14 M	152	36	225	115	36.2	60.3	18.3	1.67	
41	27	70 M	167	39	295	121	38.2	63.2	19.3	1.66	
42	28	63 F	147	63	245	107	35.1	57.9	14.3	1.65	
43	42	46 M	162	68	285	146	47.9	76.9	20.8	1.61	
44	55	35 M	172	44	332	215	71.6	115.4	27.6	1.61	
45	38	52 M	180	53	260	137	43.7	69.9	23.6	1.60	
46	17	52 M	167	45	365	157	47.5	74.8	34.2	1.58	
47	14	6 F	58	34	30	18	6.0	9.5	2.5	1.58	
48	5	45 F	150	35	325	184	60.5	95.2	28.7	1.58	
49	45	25 F	165	50	330	167	58.5	91.1	17.5	1.56	
50	12	64 M	175	53	320	166	56.5	88.3	21.6	1.56	
51	39	55 M	185	57	360	186	64.1	96.8	25.3	1.51	
52	60	18 M	170	37	207	132	45.3	67.9	19.2	1.51	
53	50	34 F	160	43	265	145	50.4	73.7	20.7	1.46	
54	20	43 F	165	67	400	199	71.2	101.6	26.2	1.45	
55	2	49 M	163	58	340	170	61.9	87.9	20.0	1.42	
56	43	61 M	166	41	260	125	45.1	62.3	17.7	1.38	
57	56	43 M	172	52	326	208	80.7	96.3	30.7	1.19	
58	23	21 M	167	48	580	261	107.3	120.2	33.3	1.12	
59	16	32 F	154	65	350	149	68.2	66.0	14.7	0.96	
Not used in averages. 60	68A	40 M	189	705	745	440	235.5	142.5	63	0.62	
Not used in averages because curves showed over-shooting. ..	15	67 M	155	51	295	138	44.3	66.3	26.2	1.49	

TABLE II.—*Pathologic findings (concluded).*

Kidney Lesions.	B. P. Systolic.	B. P. Diastolic.	General Remarks.	Valvular Defects.	Myocardial Lesions.
gms. L. 230 gms. Cl. sw. changes.	110	75	Acute lymphatic leukemia.	None.	Vessels engorged with immature lymphocytes. Cl. sw. Lymph. in myo.
gms. L. 220 gms. Vessels scl. h scarring. Chr. nephritis.	150	67	Emphysema. Pleural effusion. Toe pneumonia. Atheromatous dil. aorta.	Dil. aorta. Rel. aorta insuff.	CT. increase. Subendo. mononuc. infiltration.
gms. L. 130 gms. Sl. inc. CT. inc. change in vessels.	140	70	Appendiceal abscess. Gen. peritonitis.	None.	Mod. CT. inc. Cloudy swelling. Sclerosis. Endocard. thickened.
gms. L. 160 gms. Few scl. eruli.	140 125	85 100	Thrombosis of cavernous sinus. Some atheroma of aorta.	None.	Cloudy sw. Sl. CT inc. Dif. mononuc. infl.
L. 565 gms. comb. Rt. hydro-sclerosis. Cl. sw.	115	63	Pernicious anemia. Broncho-pneumonia.	None.	Sl. cl. sw. subepicard. mono and CT infiltration.
L. 225 gms. comb. Cl. sw. of vessels.	96	50	Adenocarc. of stomach. Gen. arteriosclerosis.	Fibrosis of valves rel. M. & T regurg.	Foci of polys. CT inc. Local thick. in endoc.
L. 140 gms. each. Adv. deg. of ar. ep. Chr. nephritis.	142	90	Glioma of VIII nerv. Aorta atheromatous.	None.	Ext. fatty atrophy. Adv. Cl. sw. CT. inc. suppurative process near e. ear.
gms. L. 32.5 gms. Ac. glom. interest. neph.	95	75	Erysipelas. Congenital syphilis.	None.	Dif. CT increase. Fatty deg. inf. Cl. sw.
gms. L. 135 gms. Cloudy ng.	120	50	Emphysema. Broncho-pneu. Inf. Uterine myomata. Gen. arterio sclerosis.	None.	CT. increase. Areas of calcif. Cl. sw. Smp. nec. Brown atrophy. Scl.
gms. L. 150 gms. Acute neph.	115	75	Cardiac failure. Gen. anasarca. Hydrothorax. Ascites.	Subacute M and aortic endocarditis. S. Virid. A. M & T regurg.	Abscesses. Marked CT increase. Cl. sw. Simple and coag. nec. Fat. deg. inf.
gms. L. 175 gms. Bilat. hydro-sclerosis.	145	85	Aur. fib. bil. Vent. extrasystoles. Card. failure. Old syphilis. Scl. of coronaries.	Fibrosis of aortic segments. No regurgitation.	Fibrosis. Fatty atrophy and deg. inf. Acute cong. Cloudy swelling.
gms. L. 240 gms. Congestion.	140 115	82 70	Adenocarc. of stomach. Trans. Aur. fib. Chr. adh. pericarditis.	None.	Chr. epicarditis. CT increase. Cloudy swelling.
L. 250 gms. comb. Cloudy ng.	110	80	Dementia praecox. Parf. of esophagus. Pneumothor. Broncho-pneu. Sl. atheroma of aorta.	None.	Cloudy swelling. Slight CT. inc. Some thickening of the vessels.
swelling. Edema of inter-tissue.	133	70	Broncho-pneumonia. Aorta normal.	None.	Slight CT increase about vessels. No other changes present.
gms. L. 150 gms. Cloudy ng.	105	68	Alkemic leukemia. Sivera. Anemia. Ascites. Atheromatous aorta.	None.	Marked fatty deg. Inf. Fatty atrophy. Leukemic inf. Increase in CT.
ed parenchymatous degenera-	120	90	Gen. arterio. Sclerosis. Sph. aortitis e. Aneurysm.	None.	Fibrosis. Congestion. Perivascular inf. of mononuc. Brown atrophy nodular thick. of endo.
520 gms. comb. Acute tubu-	120	90	Gen. arterio sclerosis. Pulm. and adv. aortic sclerosis.	Scl. and calc. of aortic V. flaps. No regurg.	Sclerosis of vessels. Mod. fibrous tissue inc. some calcification.
L. 450 gms. comb. Marked stion.	140 125	80 80	Anthraxis. Emphysema. Massive pulm. thc. Gen. millary thc. Slight atheroma of aorta.	Tricusp. regurg. by hydrostatic test.	Tubercles in myocardium. Cloudy Swelling. Smp. necrosis.
passive congestion. Tubular ar. epith. swollen and gan.	115 140	50 60	Aur. fib. cardiac failure. Pericardial adhesions.	Mitral stenosis and regurg. Aortic regurg. Few sml vegetations.	Some fibrosis. Cloudy swelling. Hypertrophy of myocardial cells.
a of lt. kidney. Rt. kidney Ac. parenchy. nephritis.	140	88	Very adv. Pulmonary emphysema (chr.).	None.	Cloudy swelling. Some diffuse leuc. infl. Blood vessels thicken.
ion. Old infarction.	100	80	Cardiac failure with congestion and oedema ac. fibinous pleurisy. Pulmonary infarcts.	Mitral stenosis and regurg. tricuspid regurg. Fenestration of pulmonary cups.	
gms. L. 160 gms. Congestion. nuc. infiltration. Scars. ic nephritis.	90	65	Lobar pneumonia. Hydrothorax and hydro-pericard. Cirrhosis of liver.	None.	Cloudy swelling. Some inc. in Connective tissue. Sclerosis of vessels.

TABLE III.—*Electrocardiographic measurements and findings.*

No.	Case No.	Age, Sex.	Vent. Wt. Gms.	L/R	Index	P1	P2	P3	Q1	Q2	Q3	R1	R2	R3
1	58	39 M	387	3.83	33.0**	t	1.0	1.0	1.0	0.0	0.0	19.0	11.0	5.0
2	22	14 F	177	3.59	5.5	0.5	1.0	0.5	0.0	t	1.0	6.0*	7.0	2.0*
3	19	47 M	224	2.86	-4.0	t	0.5	0.5	0.0	1.0	1.5	4.0	9.0	6.5
4	48	35M	351	2.82	3.5*	1.0	t	-0.5	0.0	0.5	1.0	11.0	9.0	6.0*
5	3	32 M	391	2.72	14.0** 2.5	1.0 0.5	1.0 1.5	±t 1.0	0.0 0.0	0.0 0.0	0.0 0.0	8.0 4.0	6.0 6.0	0.5 3.0
6	47	38 M	334	2.62	16.0*	1.5	3.0	2.0	0.0	0.0	0.0	11.0	13.0	6.0
7	37	60 F	91	2.57	2.5	t	t	t	0.0	0.5	1.0	4.0	5.0	1.5*
8	1	78 F	158	2.53	7.5*	t	1.5*	1.5*	t	0.0	0.0	3.5*	8.5	7.0
9	33	60 F	194	2.50	9.0	t	1.0	0.5	0.0	0.0	0.0	6.5	7.0	0.0
10	36	76 M	130	2.48	10.0** 6.5*	t t	0.5 0.5	0.5 1.0	0.5 0.5	0.0 0.0	0.0 0.0	6.0 5.0	1.0* 3.0*	1.0 1.0
11	7	48 M	427	2.46	17.8**	0.5	1.0	-0.5	0.0	0.0	0.0	13.0	6.0	1.2
12	9	44 M	163	2.46	-3.0	1.0	2.0	1.0	0.0	0.0	0.5	6.0	8.0	8.0
13	24	48 F	101	2.36	-6.0	t	1.0	1.0	0.0	0.0	t	1.0	9.0	7.0
14	6	57 M	181	2.25	7.0* -3.5	t t	1.5 2.0	1.0 2.0	0.5 t	0.0 2.0	0.0 2.0	4.5 3.5	6.0 10.0	4.0 7.0
15	10	59 M	146	2.25	-3.5	t	0.5	0.5	0.0	1.0	1.0	7.0	11.0	8.5
16	31	75 F	128	2.20	0.0	t	0.5	0.5	0.0	1.0	1.0	7.0	13.0	7.0
17	40	37 F	276	2.20	7.0*	t*	1.0	1.0	0.0	0.0	0.0	4.0*	4.5	3.0*
18	41	49 M	163	2.14	12.5**	1.0	1.5	1.0	t	0.0	0.0	6.0	3.0	2.5
19	51	58 M	140	2.02	4.0	1.0	2.0	1.0	t	0.5	0.5	6.0	5.0	1.5
20	52	68 M	178	1.99	7.5	0.6	0.6	-t	0.0	0.0	0.0	5.5	5.5	0.0
21	26	17 F	157	1.97	1.0	0.5	1.0	0.5	0.5	0.5	1.0	1.5	7.0	4.5
22	13	70 F	160	1.96	17.0**	1.5	2.0	1.0	t	1.0	0.0	10.0	5.0	0.0
23	44	41 F	138	1.96	0.5	0.5	1.5	0.5	0.0	1.0	1.0	7.5	14.0	6.0
24	29	71 M	323	1.94	9.5**	t	1.0	0.5	0.0	0.0	0.0	6.0	3.0	1.5
25	18	63 M	372	1.94	16.5**	t	1.0	1.0	t	0.0	0.0	9.5	3.0	1.5
26	8	17 M	119	1.93	7.0	0.5	1.5	1.0	0.0	0.0	t	8.0	12.0	4.0
27	54	56 F	119	1.92	5.5	0.5	1.5	1.0	t	0.0	0.0	8.5	15.0	6.5
28	49	33 F	185	1.83	1.5	0.5	1.0	t	0.0	0.0	t	6.5	9.5	4.0
29	57	26 F	107	1.82	-13.0*	0.5	1.0	t	0.0	0.5	2.0	2.0	6.5	10.0
30	35	33 F	152	1.80	8.0	t	2.0	1.5	0.0	0.0	0.0	7.0	8.0	2.0
31	11	63 M	111	1.80	6.0 1.0	t t	1.0 1.0	t t	1.0 1.0	1.0 1.0	1.5 0.0	8.0 6.0	9.0 12.0	2.0 5.0
32	30	34 M	173	1.78	-4.0	1.0	2.5	2.0	0.5	1.0	t	4.0	11.0	8.0
33	46	54 F	87	1.75	5.0	1.0	0.5*	t	1.0	1.5	1.5	8.0	10.0	2.0
34	34	38 M	159	1.71	0.5	t	0.5	0.5	0.5	t	t	2.5	3.5	2.0*
35	21	16 M	139	1.70	-18.5**	0.5	1.5	1.0	0.0	0.0	0.0	2.0	11.0	15.5
36	25	46 M	147	1.70	-6.5	t	3.0	2.5	0.0	0.0	0.0	4.0	12.0*	9.5*
37	32	31 M	193	1.70	9.0 5.5	0.5 0.5	1.5 1.0	1.0 1.0	1.0 0.5	1.0 0.5	0.0 0.0	11.0 7.0	12.0 8.0	2.0 1.5

TABLE III.—*Electrocardiographic measurements and findings.*

<i>SS</i>	<i>T1</i>	<i>T2</i>	<i>T3</i>	Remarks.	Interval between E. C. G. and death in days.
19 0	-3.0	±1.0	2.0	QRS normal. Lead I and III diphasic. Suggests right branch defect. Chr. myocarditis and nephritis.	1
2.0	t	-2.0	-2.0	No electrocardiographic evidence of myocarditis. Chronic nephritis.	20
0.5	1.5	1.5	t	Sub-acute bacterial endocarditis. Aortitis.	38
0.0	-1.5	-1.5	-1.0	Digitalis administered. Occasional extrasystoles. Chr. nephritis.	7
6.5	0.5	2.0	1.0	Pericarditis. Chr. nephritis.	10
2.0	0.5	2.5	2.0		
11.0	t	2.5	2.5	Chronic nephritis.	3
0.0	t	-t	-t	Small complexes. Cancer of hepatic duct.	60
11.0	-t	1.5	1.0	Aneurysm. Displaced heart. Cancer of bladder.	5
2.5	-t	t	1.0	P-R interval slightly increased (.23 seconds). Pyonephrosis. diabetes.	20
5.0	t	t	t	Small complexes. Thrombosis of portal vein. Ascites. Interval.	6
2.5	-t	-0.5	-t		
7.0*	-0.5	-0.5	-t	Vegetative endocarditis. Aortic regurgitation.	15
0.0	1.0	2.0	1.5	Purpura hemorrhagica. Pneumonia.	0
0.0	0.5	1.5	1.5	Pernicious anemia.	1
6.5	t	3.0	3.0	Cancer of prostate.	20
0.0	t	4.5	4.5		
0.0	1.5	3.0	3.0	Diabetes mellitus. Syphilis.	8
0.0	-t	-1.5	-1.0	Endarteritis obliterans. Cancer of cervix uteri.	21
6.0	t	-1.0	-1.0	Mural thrombus in lt. ventricle. Ac. Endocarditis. Ileocolitis (Paratyp. B).	12
9.0	1.0	2.5	2.0	Hemochromatosis. Cirrhosis of liver. Cancer of liver. Arterio sclerosis.	1
t	t	t	-t	Cancer of esophagus.	0
2.0	1.8	1.0	-1.0	Cancer of stomach.	0
1.5	2.0	2.0	t	Rheumatic aortic and mitral regurg.	4
7.0	t	t	t	Arterio sclerosis. Sclerosis of mitral ring. Diabetes mellitus.	1
t	t	t	t	General peritonitis.	5
6.0	-t	-1.0	t	Arterio sclerosis. Chronic nephritis. Digitalis administered.	6
10.5*	t	t	t	Arterio sclerosis. Chronic nephritis. Digitalis administered. P-R .22 sec.	6
5.0*	t	2.0	1.5	Epidemic encephalitis.	30
3.5	t	-t	-t	Cancer of stomach. Arterio sclerosis.	34
t	-0.5	t	t	Post-partum thrombosis of pulmonary artery.	14
0.0	1.0	t	-0.5	Cancer of right lung. Severe secondary anemia.	49
3.0	0.5	0.5	t	Interventricular septum defect. Pyelo-nephritis. Uremia.	43
0.0	t	t	t	Cancer of esophagus and rectum. Arterio sclerosis.	44
0.0	t	t	t		18
0.0	2.0	5.0	3.0	Mastoiditis. Brain abscess. Meningitis.	3
0.0	1.5	1.0	t	Cancer of breast. Tachycardia.	6
0.0	t	0.5	0.5	Cancer of stomach. Marked ascites. Very small complexes.	0
2.5	2.0	4.0	3.5	Cerebro-spinal meningitis.	3
0.0	t	2.0	-2.0	Innominate aneurysm. Aortitis.	9
0.0	-0.5	-1.0	-t	Pernicious anemia. Cardiac failure.	81
0.0	0.5	1.0	0.5		38

TABLE III. —*Electrocardiographic measurements and findings (continued).*

No.	Case No.	Age, Sex	Vent. Wt., G	L/R	Index	P1	P2	P3	Q1	Q2	Q3	R1	R2	R3
38	59	19 M	169	1.70	-11 0	0 5	1.8	1 0	0 0	1 0	2 0	3 0	12 0	12 0
39	53	22 M	200	1.69	-2 5	0.5	1.0	t	0 0	0 0	2 0	7 0	9 0	4 5
40	4	14 M	115	1.67	2 0	0 5	0.5	0 5	t	t	0 0	9 0	8 5	5 0*
41	27	70 M	121	1.66	-2.5	t	1.0	1 0	0 0	0 0	t	3 0	7 0	7 0
42	28	63 F	107	1.65	18 0**	1 0	1 5	0 5	0 5	0 0	0 0	12 0	3 0	1 0
43	42	46 M	146	1 61	-1.0	0.5	1 0	≈0 5	0 0	0 0	1 0	6 0	7 0	5 0*
44	55	35 M	215	1 61	-3.5	t	1.2	1 0	t	0.5	t	4 0	12 0	7 5
45	38	52 M	137	1.69	0 0	t	1 5	1 5	0 0	t	0 0	4 0	8 0	4 0
46	17	52 M	157	1.58	-4 0	0 0	1 0	1 0	0 0	t	t	3 0	11 0	7 0
47	14	6 F	18	1.58	-4.5	1 0	1 5	0 5	t	1 0	2 0	5 5	12 0	7 0
48	5	45 F	184	1.58	6 5	t	2 0	1 5	0 0	0 0	t	7 0	7 0	1 5*
49	45	25 F	167	1 56	1 0	1 0	1 0	t	1 0	0 5	0 0	5.5	9 0	5 0
50	12	64 M	166	1.56	-3 0	Aur. Fibril.			0 0	1 0	1 0	3 0	8 0	6 0
51	39	55 M	186	1 51	-5 0	0.5	1 5	1 0	0 0	0 0	t	1 5	7.5	7 0
52	60	18 M	132	1 51	6 5	0.5	1 5	1 0	t	0 0	t	5 0	8 0	4 0
53	50	34 F	145	1 46	0 5	1 0	1 0	t	t	1 5	1 0	7 0	12 0	8 0
54	20	43 F	199	1 45	26.5**	t	1 5	1 5	2 0	0 0	0 0	19 0	12 0	3 0
55	2	49 M	170	1 42	2.5 3.5	±t 1 0	±t* 2 0	±t* 1 5	t 0.5	0 0 0 0	0 0 0 0	3 0 4 0	3 0 3 0	t 0 5
56	43	61 M	125	1 38	2 5	t	1 0	1 0	1 0	1 0	0 0	4 0	7 0	3 0
57	56	43 M	208	1 19	-8 0	0 5	2.5	2 0	0 0	1 0	1 0	3 0	10 0	9 0
58	23	21 M	261	1 12	-7 0	Aur. Fibril.			0 0	0 0	t	4 0	12 0	11 0
59	16	32 F	149	0 96	-6 0	t	3 0	2.5	0 0	1 0	2 0	2.5	9 0	8 0
60	68A	49 M	440	0 62	-16 0	2 0	1 5	1 0	t	0 0	0 0	2.5	5 0	11 0

* Electrocardiographic measurements so marked indicate that the corresponding deflections were notched.

** Indices so marked indicate that corresponding electrocardiograms show certain signs of right or left ventricular preponderance as described in text.

Italic figures indicate that the corresponding electrocardiographic measurements exceed the normal limits of Lewis and Gilder.

TABLE III.—*Electrocardiographic measurements and findings (continued).*

<i>SS</i>	<i>T1</i>	<i>T2</i>	<i>T3</i>	Remarks.	Interval between E. C. G. and death in days.
0.0	1.0	3.0	2.0	Miliary tuberculosis.	6
t	1.5	1.0	t	Osteomyelitis of jaw. Leukemia.	11
0.5	3.0	3.0	1.0	Lymphatic leukemia.	36
1.5	0.5	1.5	1.0	Tbc pneumonia. Dilation of aorta.	36
7.0	±1.0	1.0*	2.0*	General peritonitis. (Chr.)	1
0.5	1.5	t	-1.0	Thrombosis of cavernous sinus.	5
0.0	t	-1.0	-0.8	Pernicious anemia	3
1.0	1.0	1.0	0.5	Cancer of stomach. Arterio-sclerosis. Severe secondary anemia.	14
2.0	1.5	2.5	1.5	Glioma of VIII nerve.	2
0.0	3.5	2.5	-1.5	Erysipelas. Congenital syphilis. QRS normal. Lead I and III diphasic.	18
1.0	1.0	1.0	t	Infected uterine myomata. Arterio-sclerosis. Secondary anemia.	12
0.5	t	1.0	0.5	Sub-acute bacterial endocarditis. Strep. Viridans. Mitral stenosis and regurg. and aortic regurgitation (old).	6
0.0	t	-1.5	-1.5	Lobar pneumonia. Arterio-sclerosis. Angina (?) Hydronephrosis.	1
0.5	-0.5	-3.0	-2.5	Cancer of stomach. Chr. adh. pericard. Transient aur. fib. Digitalis admin.	3
6.0	t	4.5	3.0	Dementia praecox. Perforation of esophagus. Pneumothorax	1
1.5	1.5	1.5	t	Bronchopneumonia	0
10.5*	1.5	1.5	-t	Aleukemic leukemia. Severe anemia.	63
0.5	1.0	1.5	t	Aortic aneurysm. Small complexes	3
1.5	2.0	2.5	t		
1.5	1.0	2.0	1.0	Perforating gastric ulcer. Arterio-sclerosis. Abscess of liver.	58
0.0	1.5	3.0	1.5	Emphysema. Anthracosis. Massive pulmonary tuberculosis.	14
3.5	?	?	?	Mitral stenosis, and regurgitation. Aortic regurgitation. (Rheumatic)	1
0.0	t	0.5	t	Very severe chronic pulmonary emphysema.	73
-1.0	2.0	-2.5	-3.0	Mitral stenosis. Cardiac failure.	3

TABLE IV.—Average values.

	a	b	c	d	e	f
	Normals.	Normals.	Normals. (Lewis.)	Normals. (L. & G.)	Cases. 2.20+	Cases. 1.80+
Average number of cases.....	16	12	11	52	15	16
Age.....	32.9	31.4			49.0	48.5
Height in gms.....	164	165			165.7	159.8
Weight in kgs.....	49.7	50.3	49.4		47.9	49.3
Heart wt. in gms.....	255	254	304		419	344
Vent. wt. in gms.....	141	146	148.7		230.6	176.0
R. vent. weight.....	42.3	45.8	44.3		51.6	50.3
L. vent. weight.....	77.0	78.6	81.4		139.9	98.9
Septal weight.....	20.9	21.6	23.1		38.5	27.7
L/R ratio.....	1.74	1.73	1.83		2.68	1.96
Vent. weight.....	.00292	.00294	.00301		.0047	.00363
Body weight.....						
Index.....	0.25	-3.3		-1.8	5.58	5.34
P1.....	0.64	0.61		0.52	0.49	0.56
P2.....	1.24	1.20		1.16	1.15	1.25
P3.....	0.66	0.58		0.81	0.82	0.69
Q1.....	0.23	0.22		0.51	0.14	0.18
Q2.....	0.44	0.59		0.73	0.36	0.34
Q3.....	0.75	0.95		0.86	0.55	0.38
R1.....	5.97	5.37		5.16	6.96	6.49
R2.....	8.89	9.52		10.32	8.17	7.87
R3.....	5.34	6.84		6.61	4.65	3.68
S1.....	1.91	2.34		2.06	0.58	0.83
S2.....	2.34	1.46		2.23	1.53	1.81
S3.....	2.03	0.49		1.73	3.83	3.39
T1.....	1.04	1.26		1.93	0.04	0.48
T2.....	1.89	1.68		2.46	0.96	0.40
T3.....	1.12	0.78		0.61	1.02	0.11

Columns of above table include:

a. Normals.

No cardiovascular disease other than slight atheroma of aorta. No chronic renal or pulmonary disease. No severe anemia. Systolic over 140.

Cases:

Numbers 18, 23, 26, 28, 29, 32, 33, 34, 35, 38, 39, 40, 42, 43, 52, and 53.

b. Normals.

Same as (a) except that four cases with distinctly abnormal electrocardiograms are excluded.

Cases:

Same as (a) except that Cases 18, 26, 42, and 52 are omitted.

c. Normals.

Lewis Cancer Controls.

See Lewis (9).

d. Normals.

Lewis and Gilder (10).

e. Cases with *L/R* ratios over 2.20.

Cases 1 to 16 inclusive.

f. Cases with *L/R* ratios between 1.80 and 2.20.

Cases 16 to 31 inclusive.

TABLE IV.—Average values.

<i>h</i>	<i>i</i>	<i>j</i>	<i>k</i>	<i>l</i>	<i>m</i>	<i>n</i>		<i>o</i>	
Cases 1.50—	Same —(54)	Cases .075+	Cases 180 +	Cases 180—	Cases A-S	Aortic Regurg. Lewis.		Mitral Stenosis. Lewis.	
	6	6	8	11	6	13	31	13	40
4		34.3	44.1	59.2	56.5				
8		164	163.1	164	162.3				
0		54	51.4	46.0	43.6	69.0		53.7	
1		530	495.3	332	302.5	711		435	
3		345	291.1	153	165.6	421.7		256.7	
6		73.3	65.2	43.3	51.4	112.7		97.3	
9		211.1	174.2	84.4	92.4	241.7		117.0	
3		59.9	50.7	25.0	21.9	63.3		42.2	
23		3.01	2.75	1.99	1.85	2.1		1.55	
.0344		.07355	.07539	.00331	.07331				
.67	-2.42	13.1	9.2	3.04	4.2		8.36		10.14
.56	0.62	0.72	0.59	0.47	0.34		1.05		1.17
.63	1.03	1.30	1.30	1.12	1.23		1.57		2.05
.46	1.45	0.67	0.94	0.75	1.19		0.94		1.15
.54	0.30	0.17	0.20	0.15	0.47		0.54		0.34
.64	0.75	0.13	0.47	0.39	0.33		0.62		0.84
.61	0.71	0.33	0.63	0.31	0.21		0.61		1.30
.21	4.03	13.7	8.9	5.32	5.25		9.31		3.05
.29	8.83	8.70	9.09	7.40	7.41		11.61		11.17
.07	6.53	3.93	4.70	3.68	3.59		5.27		11.27
.11	1.30	0.63	0.60	0.55	0.0		1.37		3.47
.43	1.33	2.08	2.06	0.68	0.33		3.57		2.09
.64	1.33	6.80	5.56	1.98	2.41		5.79		1.25
.30	1.05	-0.65	-0.39	0.65	0.43		0.87		1.47
.57	1.53	0.17	0.68	0.87	-0.05		1.40		2.49
.44	0.57	0.53	0.91	0.63	-0.03		0.65		1.16

h. Cases with *L/R* ratios between 1.50 and 1.80.

Cases 32 to 52 inclusive, omitting case 47.

i. Cases with *L/R* ratios below 1.50.

Cases 53 to 59 inclusive.

Same as (*h*) but case 54 omitted.

k. Cases with *L/R* ratios above 2.20 and Vent. Wt. Body Wt. ratio above .005.

Cases 1, 2, 4, 5, 6, and 10.

l. Cases with chronic nephritis and systolic B. P. above 180 mm mercury.

Cases 1, 2, 4, 5, 6, 14, 16, and 24.

m. Cases with chronic nephritis and systolic B. P. below 180 mm mercury.

Cases 7, 13, 19, 15, 20, 22, 24, 30, 31, 41, and 46.

n. Cases of arterio sclerosis.

Cases 10, 17, 27, 48, 50, and 56.

o. Cases with aortic regurgitation (Lewis) (8).

The pathological data are from one series of cases while the electrocardiographical data are from another series of cases.

p. Cases with mitral stenosis (Lewis) (8).

The pathological data are from one series of cases while the electrocardiographical data are from another series of cases.

TABLE V.—*Miscellaneous data.*

No.	Case No.	Vent. Wt.	L/R	L-1.8R	Index	Carter's Index	Incl. of an. axis X-ray.	Causes for displacement of the heart.
1	58	387	3.83	131.4	33.0**	-15		
5	3	391	2.72	77.9	14.0* 2.5	-16 42	28*	Fibrino-pur. Pericard. Pleural effusion lt.
4	48	351	2.82	76.0	3.5*	53	46*	
11	7	427	2.46	70.4	17.8**	1	36*	
6	47	334	2.62	63.4	16.0*	3	42*	
2	22	177	3.59	57.1	5.5	30		Right empyema
3	19	224	2.86	51.9	-4.0	76		
9	33	194	2.50	31.7	9.0	8	43*	
17	40	276	2.20	30.6	7.0*	-16		Pleural effusion
8	1	153	2.53	26.6	7.5*	-37		Aortic aneurysm
12	9	163	2.46	25.5	-3.0	68		
14	6	181	2.25	22.0	7.0* -3.5	-1 71		
10	36	130	2.43	21.6	10.0** 6.5*	-11 13	?	Ascites
7	37	91	2.57	17.0	2.5	45	?	Ascites
15	10	146	2.25	17.0	-3.5	69		
18	41	163	2.14	15.5	12.5**	-34		Ascites
13	24	101	2.36	14.6	-6.0	83		Ascites. Hydrothorax
25	18	372	1.94	14.2	16.5**	-39	40*	
16	31	123	2.20	13.4	0.0	60		
24	29	323	1.94	12.7	9.5**	-25	26*	Ascites. Hydrothorax
20	52	178	1.99	9.6	7.5	9		
19	51	140	2.02	8.9	4.0	42		
21	26	157	1.97	7.3	1.0	55	41	Ascites. Hydrothorax
22	13	160	1.96	7.0	17.0**	-13		Sero-pur. Pericard
23	44	138	1.96	6.0	0.5	59		
26	8	119	1.93	4.3	7.0	21		
27	54	119	1.92	3.9	5.5	45		
28	49	185	1.83	1.9	1.5	55	37	
29	57	107	1.82	0.7	-13.0*	107	55	Massive calc. of rt. lung
30	35	152	1.80	0.0	8.0	22	35*	
31	11	111	1.80	0.0	6.0 1.0	41 57	35	
33	46	87	1.75	-1.0	5.0	42	36	
47	14	18	1.58	-1.3	-4.5	75		
32	30	173	1.78	-1.7	-4.0	71	36	Left empyema. Lat. Scoliosis
34	34	159	1.71	-4.0	0.5	56		Ascites
35	21	139	1.70	-4.1	-13.5**	115		

TABLE V.—*Miscellaneous data.*

Capacity, cc.	Vent. Wall Thickness mm.			Interval, days.		Remarks.
	R.	L.	R.	X-Ray to death.	E. C. G. to death.	
75	13	2		1	1	Q.R.S. normal. (0.003) Ld. I and III diphasic. Suggest conduction defect in rt. branch of His-bundle. Change in E.C.G. following removal of pleural fluid (300 cc.) probably from change in position of heart. X-ray 7 ft. M.R. 6.5 cm. M.L. 14.5 cm.
.....	15	5	7	10	10	
80	16	4	6	7	7	Curves anomalous. T inversion due to digitalis (?). X-ray 7 ft. M.R. 6.5 cm. M.L. 9.5 cm.
.....	12	2	23	15	15	
123	15	3	23	3	3	X-ray 7 ft. M.R. 5.5 cm. M.L. 9.5 cm.
9	13	6		20	20	
3	10	3		33	33	E.C.G. normal. X-ray report ventricular shadow transverse. Plate lost.
60	12	3	20	20	20	
113	11	2		12	12	Curves anomalous. Atrial flutter. Heart much displaced.
.....	10	2		5	5	
.....	11	4		0	0	
.....	11	5		20 2	20 2	
65	13	2	10	6 3	6 3	Change in E.C.G. following removal of ascitic fluid 12.5 liters, with change in position of heart, in a heart which was transverse.
44	10	5	55	60	60	
.....	9	2		8	8	Curves of very low amplitude. Heart displaced. X-ray outline of heart hidden by high diaphragm.
75	12	2		1	1	
43	9	2		1	1	X-ray 7 ft. M.R. 7.5 cm. M.L. 11 cm.
.....	14	5	5	6	6	
50	9	2		21	21	X-ray 7 ft. M.R. 5.0 cm. M.L. 11.5 cm. Heart transverse.
110	12	4	6	6	6	
75	13	4	11	0	0	Curves show sl. fling due to high skin resistance.
50	12	3	3	0	0	
28	8	4	43	4	4	Curves within normal range. Dextrocardiogram precedes Levocardiogram. (?) Architecture (?) Vertical.
.....	12	7		1	1	
40	12	4		5	5	Change in curve due to change in heart's position (?).
.....	11	3.5		30	30	
.....	14	4		34	34	X-ray taken with patient in supine position.
100	8	2	1	14	14	
45	11	5	5	49	49	Curves of very low amplitude.
85	12	3	40	43	43	
.....	9	3	44	44 18	44 18	E.C.G. just outside normal range. Heart clinically and pathologically apparently normal.
45	8	1	19	6	6	
.....	5	2		18	18	Curves of very low amplitude.
75	9	3	0	3	3	
24	9	2		0	0	E.C.G. just outside normal range. Heart clinically and pathologically apparently normal.
55	10	3		3	3	

TABLE V.—*Miscellaneous data (continued).*

No.	Case No.	Vent. Wt.	L/R	L-1.8R	Index	Carter's Index	Incl. of a.n. axis X-ray.	Causes for displacement of the heart.
36	25	147	1.70	-4.5	-6.5	77	49*	Aneurysm.....
38	59	160	1.70	-4.9	-11.0	86	43	
40	4	115	1.67	-4.9	2.0	54		
42	28	107	1.65	-5.3	18.0**	0		
41	27	121	1.66	-5.6	-2.5	70		Pleural effusion.....
37	32	193	1.70	-5.7	9.0 5.5	38 40		
39	53	200	1.69	-7.2	-2.5	73		Right empyema.....
45	38	137	1.60	-8.7	0.0	60		
42	42	146	1.61	-9.5	-1.0	64		
46	17	157	1.58	-10.7	-4.0	81		
50	12	166	1.56	-13.5	-3.0	71		
44	55	215	1.61	-13.6	-3.5	70		
52	60	132	1.52	-13.7	6.5	3	45	Pneumothorax.....
48	5	184	1.58	-13.8	6.5	39		Empyema.....
49	45	167	1.56	-13.9	1.0	57	40	Hydrothorax. Ascites.....
53	50	145	1.46	-17.0	0.5	7		Massive broncho-pneumonia.....
51	39	186	1.51	-18.4	-5.0	80	55*	
56	43	125	1.38	-18.9	2.5	45	37	Sub-diaphragmatic abscess.....
55	2	170	1.42	-23.6	2.5 3.5	16 7	25	Aortic aneurysm.....
54	20	199	1.45	-26.4	26.5**	7		Hydrothorax. Ascites. Tympanites.....
57	56	208	1.19	-43.7	-8.0	85	46	Chr. pulmonary tbc.....
59	16	149	0.96	-57.0	-6.0	79		Pulm. emphysema.....
58	23	261	1.12	-73.0	-7.0	87	40*	
Not in the averages.								
60	68A	440	0.62	-175.6	-16.0	8		Hydrothorax, pneumothorax. Heart rotated. Rt. vent. alone presented anteriorly.

X-Ray measurements marked with the sign (*) were made from tele-roentgenograms (seven foot heart plates).

Other signs (*) or (**) used with indices indicate that corresponding electrocardiograms show one or both signs of preponderance as described.

TABLE V.—*Miscellaneous data (continued).*

Capacity, .	Vent. Wall Thickness mm.		Interval, days.		Remarks.
	R.	L.	X-Ray to death.	E. C. G. to death.	
54	9	2	12	9	X-ray 7 ft. <i>M.R.</i> 4.5 cm. <i>M.L.</i> 7.0 cm.
115	9	2		6	X-ray shows vertical heart.
.....	7	2		3	
44	8	2		1	
92	5	3		36	
100	10	3		81 38	
150	11	3		11	
90	8	3		14	
85	14	2		5	
.....	9	4		2	
.....	12	3		1	
95	16	5		3	
30	11	4	1	1	Curves taken after pneumothorax developed.
.....	9	3		12	
75	7	4	6	6	X-ray reported very large heart. No measurements rec.
100	9	2.5		0	
50	13	4	3	3	Inversion of <i>T</i> wave due to digitalis.
75	9	2	40	58	
.....	11	4	655	3 647	X-ray plate shows an enormous aneurysm and a much enlarged transverse heart.
.....	11	4		63	
75	20	8	19	14	
.....	10	6		73	
92	10	6	58	1	X-ray 7 ft. <i>M.R.</i> 6.0 cm. <i>M.L.</i> 11.5 cm.
120	15	10		3	

TABLE VI.—*Comparison of the electrocardiogram and L-1.8R value in cases in which the heart was greatly hypertrophied; cases of Lewis and of Cotton included.*

Case.	Ventricular weight.	Index.	L/R.	L-1.8 R.
1	387	33 0**	3 83	131 4
5	391	14 0** 2 5	2 72	77 9
4	351	3 5*	2 82	76 0
C. 4	421	17 5**	2 55	74 0
11	427	17 8**	2 46	70 4
C. 3	404	29 5**	2 63	70 0
6	324	16 0*	2 62	63 4
L. 1	270 5	19 0**	2 60	52 0
14	276	7 0*	2 20	30 6
L. 2	343	19 0**	2 04	23 0
C. 5	350 5	-10 0	1 97	16 0
25	372	16 5**	1 94	14 2
24	323	9 5**	1 94	12 7
L. 4	308 5	8 0	1 94	12 5
L. 3	416 5	12 5**	1 87	9 0
L. 5	297	4 5*	1 82	2 0
L. 7	292	-7 0	1 60	-19 0
C. 6	333	9 0**	1 30	-24 9
C. 2	201 9	-5 5	1 30	-33 2
L. 6	285	1 5*	1 38	-42 0
58	231	-7 0	1 12	-73 0
C. 1	269	-6 0**	0 82	-116 9
L. 8	287	-15 5**	0 82	-125 0
I. 9	256	-77 0**	0 41	-223 0

C.—Cotton.

L.—Lewis.

TABLE VII.—Effect of respiration on the normal electrocardiogram.

Case N.	P (N)*	P (I)	P (E)	Q (N)	Q (I)	Q (E)	R (N)	R (I)	R (E)	S (N)	S (I)	S (E)	T (N)	T (I)	T (E)
551-L1	0.8	0.6	1.0	0.5	0.0	1.0	11.0	4.5	13.0	1.7	1.0	2.0	3.0	1.7	4.0
551-L2	t	-t	t	0.0	0.0	0.0	14.0	15.0	11.0	2.5	3.0	2.5	4.0	4.0	4.0
551-L3	t	-1.0	-0.8	0.0	0.0	0.0	4.0	9.5	1.2	2.0	2.0	5.5	1.8	2.5	0.8
Inde							7.3	-4.0	15.3**						t
520-L1	t	t	t	0.0	0.0	t	5.0	1.8	7.0	0.0	0.0	0.0	1.0	t	1.1
520-L2	0.7	t	0.7	1.2	1.0	0.8	15.0	17.0	13.0	1.5	2.5	1.2	3.2	2.7	2.7
520-L3	t	t	t	1.0	1.0	0.5	9.0	13.0	6.0	2.0	2.5	2.5	3.0	2.0	2.0
Inde							-2.0	-8.7	3.5						
527-L1	t	t	0.5	0.0	0.0	0.0	7.0	3.0	9.0	1.5	1.5	1.5	1.5	1.8	2.0
527-L2	2.5	2.2	2.7	0.0	0.0	0.0	13.0	15.0	10.0	0.0	t	1.2	2.0	2.0	2.0
527-L3	1.8	t	1.8	0.0	0.0	0.0	9.0	12.0	t	0.0	0.0	4.0	0.8	0.8	0.5
Index							-3.5	-10.5	11.4						
632-L1	t	t	0.5	0.0	0.0	0.0	2.0	1.0	2.1	2.5	1.0	3.0	0.8	0.5	0.9
632-L2	1.0	1.5	1.7	0.9	0.0	0.4	16.4	13.0	15.0	1.4	2.6	1.7	1.8	0.9	2.3
632-L3	1.1	1.5	0.9	1.1	t	0.9	16.4	12.7	17.3	0.0	1.8	0.0	0.9	0.5	1.1
Index							-16.9	-10.9	-18.9						
630-L1	t	t	t	0.0	0.0	0.0	1.5	0.6	2.0	2.2	1.5	3.0	2.0	1.2	3.0
630-L2	1.8	2.2	1.5	2.0	1.7	2.0	17.0	14.5	17.0	0.0	0.0	0.0	7.0	5.0	6.5
630-L3	1.8	2.0	1.2	3.0	2.2	3.0	16.5	15.0	16.0	0.0	0.0	0.0	5.0	4.0	3.0
Index							-17.2	-15.9	-17.0						

*N.—Normal breathing.

I.—Deep inspiration.

E.—Forced expiration.

t.—A trace.

Courtesy of Dr. Frank N. Wilson.

LEGENDS FOR FIGURES.

- Fig. 1. A. The electrocardiogram of a case of pulmonary stenosis. Note the great right ventricular preponderance and that leads I and III are not diphasic. B. The electrocardiogram of a case of aortic insufficiency. Note the great left ventricular preponderance and that leads I and III are diphasic.
- Fig. 2. A diagram illustrating the manner in which right and left ventricles and the septum are separated in the Lewis Method. Courtesy of Sir Thomas Lewis; Personal Communication to Dr. Frank N. Wilson.
- Fig. 3. A. A similar diagram illustrating the manner in which right and left ventricles and septum are separated in our Method A. (discarded)
B. A similar diagram illustrating the manner in which right and left ventricles are separated through the mid septum by our Method B (satisfactory). Note that Lewis Method can be subsequently applied.
- Fig. 4. Photographs of sections of hearts.
(A) showing guiding line through center of septum
(B) showing transverse sections which have been divided into right and left parts by our Method B.
(C) showing transverse sections and divisions along midseptal line by our Method B.
- Fig. 5. The effect of forced respiration upon the form of an electrocardiogram which indicated slight left ventricular preponderance. Lead I-above, Lead II-middle, and Lead III-above. The left end of the figure shows the form of the curve when the breath was held in deep inspiration; the gradual change in the form of the curve was the result of expiration.
- Fig. 6. The effect of forced breathing upon the form of Lead III of a curve indicative of definite left ventricular preponderance. Breath held in deep inspiration (left end of curve) and gradually expired. Note the appearance of a deep notch in QRS due to a change in the direction of rotation of the electrical axis.
- Fig. 7. A normal electrocardiogram which suggests a transversely placed heart.
- Fig. 8. A normal electrocardiogram which suggests that the right ventricle received the excitation wave in advance of the left.

FIGURE I

A

B

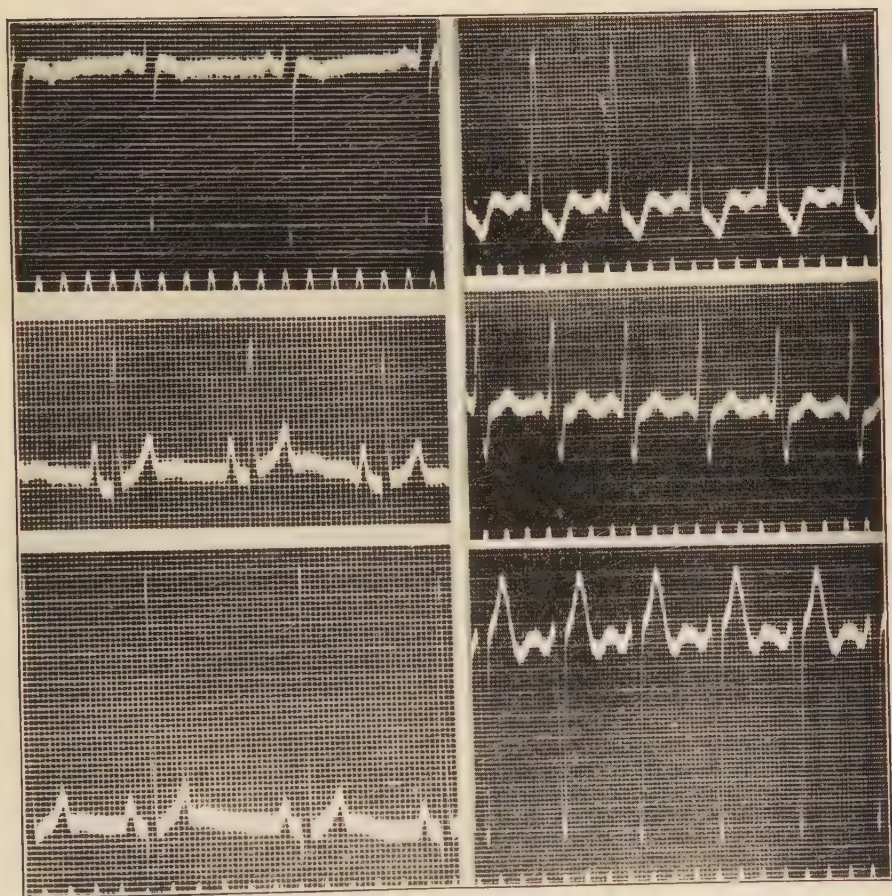
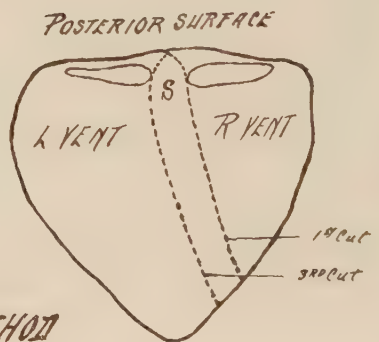
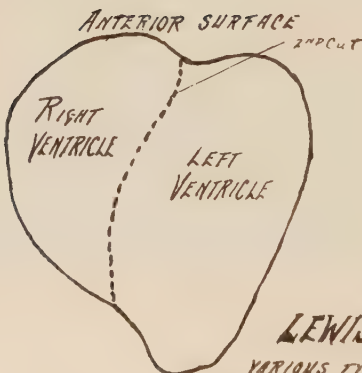
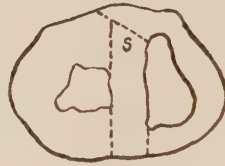
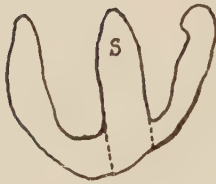
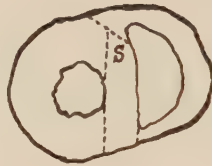
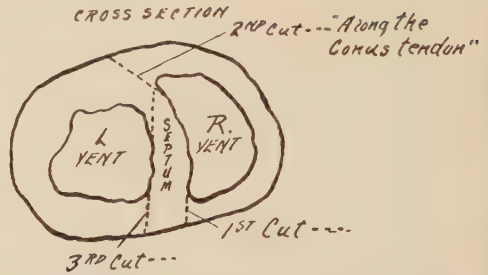
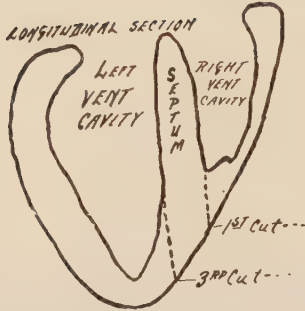
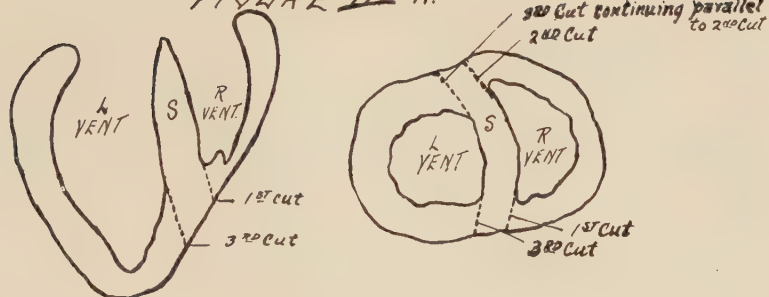


FIGURE II



LEWIS' METHOD
IN
VARIOUS TYPES OF HEARTS
FROM DISSECTIONS SENT TO DR FRANK M. WILSON
BY SIR THOMAS LEWIS

FIGURE III A.



METHOD A

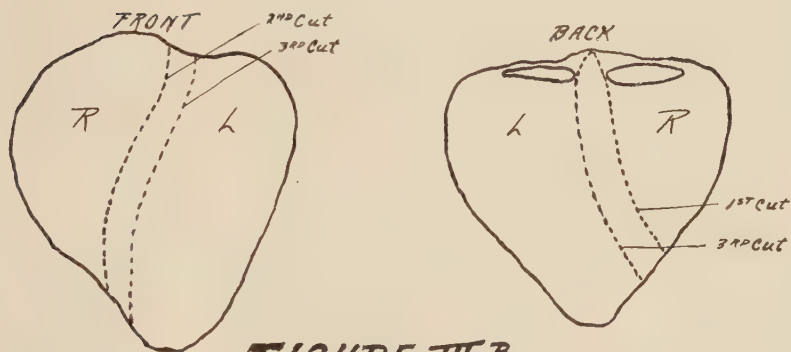
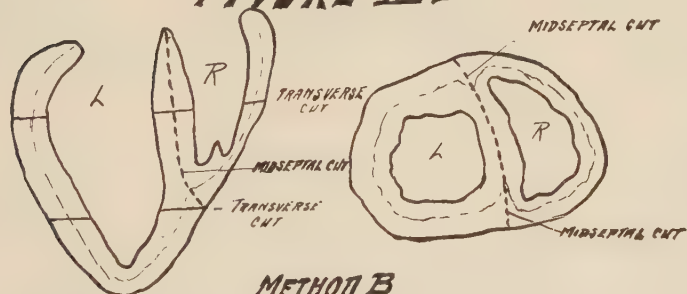


FIGURE III B



METHOD B

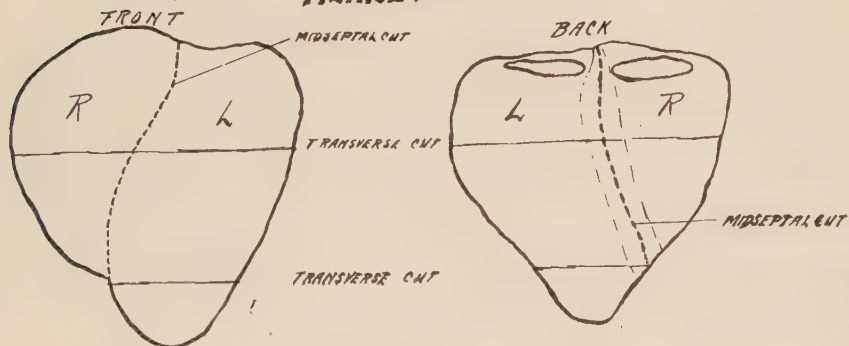
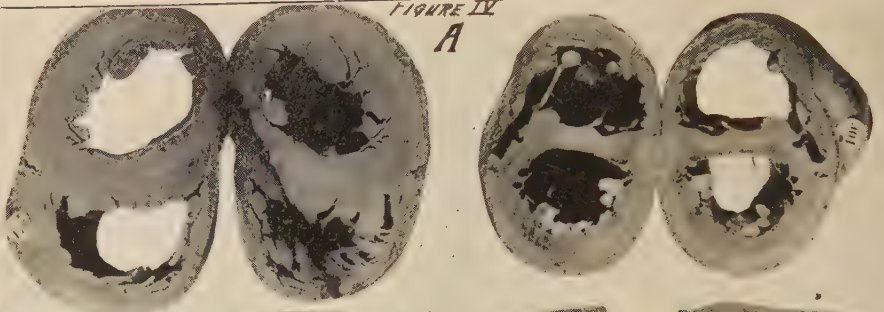
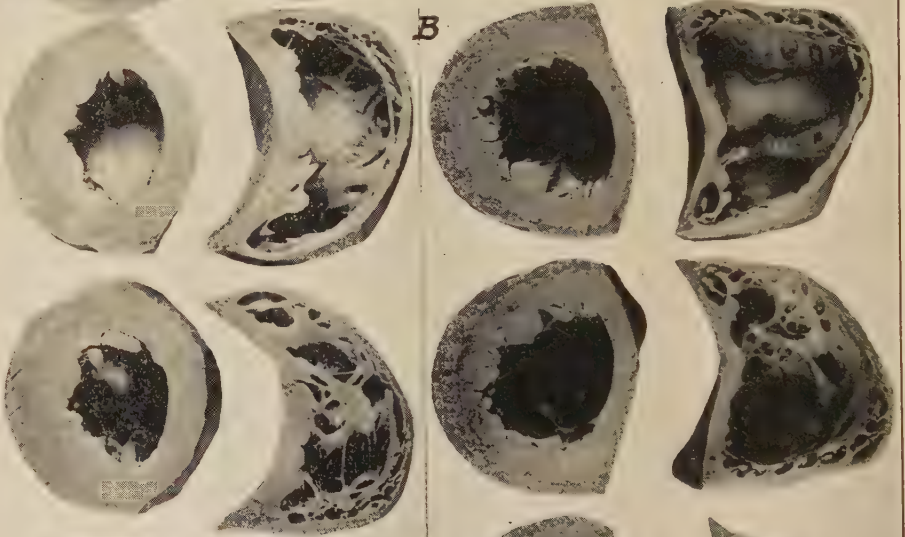


FIGURE IV

A



B



C



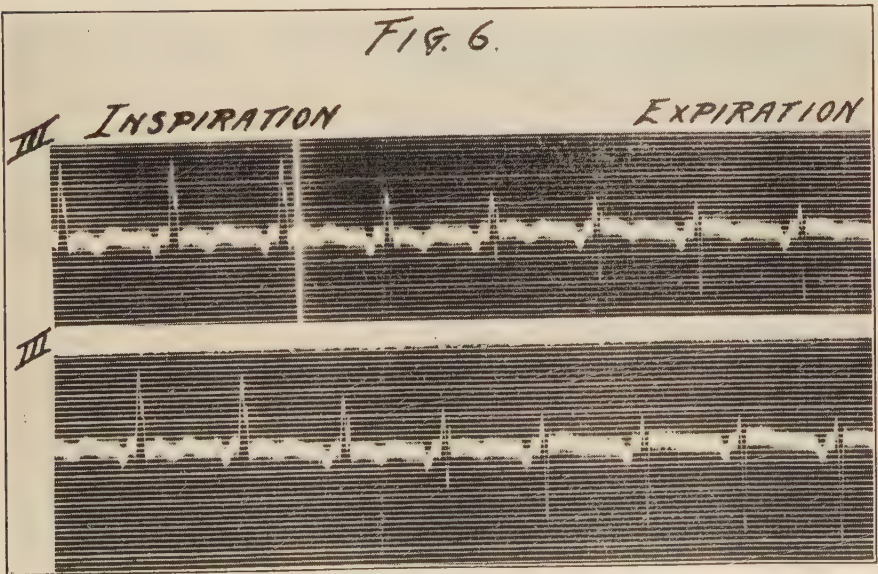
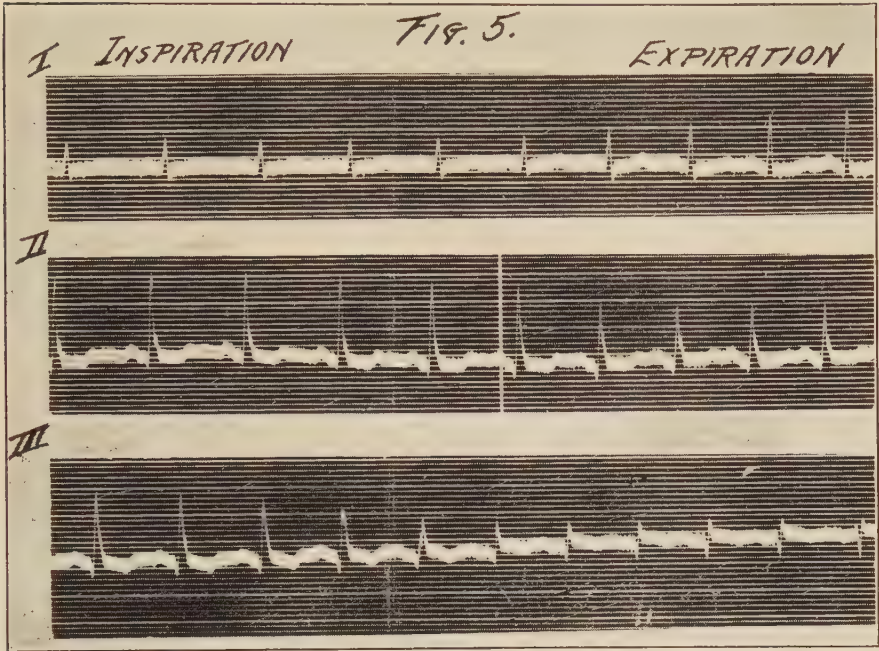


FIGURE 7

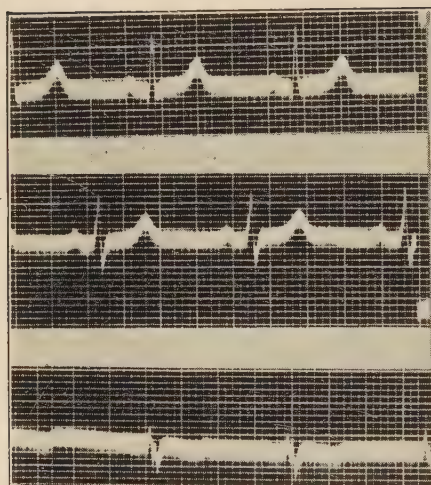
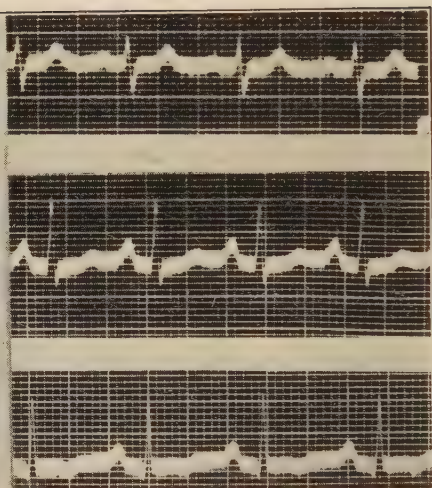


FIGURE 8



APPENDIX

CASE HISTORIES *

CASE 1—(58-68)

A man aged 39 years, entered the hospital with the complaints of severe headache, shortness of breath, and a reduction in the amount of urine passed.

The first indication that he had had of the presence of an abnormality had been the report of the finding of albumin in his urine, in about 1905. He had been told by the doctor not to worry over the finding, even though albumin had been found to be present on each analysis year after year. He had been constantly troubled with nocturia throughout this period. The symptoms which had caused him to come to the hospital, for relief, had been present for a year and had been gradually increasing in severity. He had suffered from rather severe frontal and occipital headaches and from extreme nervousness. He had taken a three week's vacation trip, as a rest, some months before admission, but his symptoms had progressed. He had noted an increasing shortness of breath and a failing of vision. Baths and mineral waters had not relieved his symptoms. He had not limited his diet, but had taken the usual amounts of foods until nine weeks before admission when his appetite had begun to fail. Four weeks later he had begun to limit his diet to milk, toast, and eggs. He had been in bed most of the time for a week, and had received digitalis for his dyspnoea and hypodermic injections (probably of morphine) for his headaches.

In his PAST HISTORY he had had a number of infections which may have played some part in the production of the disease process, which was incapacitating him. Besides the common diseases he had had scarlet fever during childhood and diphtheria at the age of twelve years, but no complications had been noted with any of these infections. At the age of twenty-three years he had been sick for nine months with very severe attack of typhoid fever.

His MARITAL HISTORY was of no great significance. His wife had had one miscarriage at seven months. The second pregnancy was, however, uneventful. The mother and child were living and well.

The FAMILY HISTORY was of interest only in that a niece had died of Bright's disease.

The PHYSICAL EXAMINATION revealed some strikingly abnormal findings. The patient was orthopnoeic and quite pale and pasty. He was groaning and holding his hands to his forehead. The breath was very heavy and slightly urinous. The fundus of each eye, showed tortuous sclerotic arteries, numerous large white patches, and a few small hemorrhages.

The peripheral arteries were thickened and tortuous. The radial pulse was regular, strong, full, well sustained and of increased tension. The blood pressure was 240 mm. of mercury systolic and 160 mm. of mercury diastolic.

The heart was considerably enlarged. The apex impulse was visible and palpable in the fourth and fifth interspaces as a diffuse but forcible impact. The point of maximum intensity was 12 cm. to the left of the midsternal line. The left border of the cardiac dullness was percussed at 14 cm. from the midsternal line, while the right border was found to be 3.5 cm. to the right of the midsternal line. The aortic dullness measured 6.5 cm. across. The heart was regular. The second aortic sound was loud, sharp, and ringing. A soft blowing systolic murmur was heard in the mitral area and in the anterior part of the axilla.

The examination of the abdomen and extremities revealed no findings of significance.

The LABORATORY EXAMINATIONS showed some significant findings. The urine was found to contain a small amount of albumin and many large hyalin and coarsely granular casts were demonstrated in the sediment. The hemoglobin and red blood cell count were normal; the leucocyte count was 19,800. The blood Wasserman reaction was negative. Microchemical analyses of the blood revealed slight retention of nitrogen bodies, the non-protein nitrogen fraction amounted to 48.1 mg. per 100 c. c. of blood, the uric acid 3.32 mg. per 100 c. c. and the creatin 1.43 mg. per 100 c. c.

The electrocardiograms were of the type of left ventricular preponderance of an extreme degree.

* Abstracted in part from the records of *The Medical Service of Professor George Dock, at Barnes Hospital, Washington University School of Medicine, Saint Louis, Mo.,* and in part from the records of the *University of Michigan Hospital, Ann Arbor.*

Therapeutic procedures were of little avail. Soon after admission a venepuncture was done and 600 c. c. of blood were removed, and following this 500 c. c. of 10 per cent glucose were infused. The headache was relieved for a short time but returned and remained present in spite of icebag therapy, pyramidon, and morphine. With rest in bed and erythrol tetranitrate $\frac{1}{2}$ grain t. i. d. the blood pressure was found to be 230 systolic and 140 diastolic.

About 25 hours after admission the patient was found sitting up in bed complaining bitterly of pain. The systolic blood pressure was over 280, the highest level that the instrument at hand would register. Within five minutes the patient was unconscious and cyanotic. The respirations were very slow and stertorous. The pulse continued for a few seconds, but was extremely weak. No reflexes were obtainable. Respirations ceased except for a few infrequent gasps. The heart stopped.

The CLINICAL DIAGNOSES were: Chronic nephritis, hypertension, generalized and cerebral arteriosclerosis, chronic myocarditis, cardiac hypertrophy and chronic dilatation, albuminuric retinitis and chronic cur mia.

The PATHOLOGICAL DIAGNOSES were: Chronic diffuse nephritis, arteriosclerosis, cardiac hypertrophy and dilatation, arteriosclerosis of the cerebral vessels, and oedema of the brain. There was old caseous and calcified tuberculosis in the peri-bronchial lymph glands.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS KIDNEYS, AORTA AND HEART

KIDNEYS: The right kidney weighed 210 grams and measured 13.5x6x3 cm. The capsule was slightly adherent. The surface, stripped of the capsule, was grayish red in color and slightly, but distinctly, granular. On section, the cortical striations were found to be for the most part obscured. Numerous gray yellow opaque areas, scattered throughout the kidneys, were noted. There were many pin point yellow areas both on the surface and cut section. The left kidney weighed 215 grams and measured 14.5x6x3 cm. and was exactly similar to the right in appearance.

Microscopically, the kidneys presented the picture of a chronic diffuse nephritis. The tubules contained granular debris and hyalin casts. There were certain areas of fibrous tissue with round cell infiltration. Bowman's capsules were thickened and the glomeruli contained granular debris in varying amounts or were completely necrosed. There were some areas of focal necrosis in all parts of the kidney.

The AORTA was moderately sclerosed and inelastic with numerous yellow thickenings or raised plaques throughout, especially in the thoracic portion. The arterial branches all showed a similar sclerosis of about the same degree.

Microscopically, there was a marked intimal thickening.

The HEART was enlarged, and extended 14 cm. to the left of the midsternal line. The auricles were dilated and the left ventricle appeared to be the chamber that was most hypertrophied. The organ weighed 535 grams. The pulmonary artery was moderately sclerosed. The coronaries likewise were moderately sclerosed, but contained no thrombi or other obstructions. The heart muscle did not show any gross abnormalities.

Microscopically, there was a conspicuous increase in adipose tissue, with some fatty infiltration of the muscle near the epicardium. In the sections taken near the atrioventricular groove the muscle fibers showed a slight cloudy swelling. There was no very marked increase in the connective tissue. The blood vessels showed some thickening. In some places the fatty infiltration of the myocardium was very marked. There was also a slight mononuclear infiltration in some parts of the subepicardial fat.

CASE 2—(22-166)

A girl, age 14 years, was brought to the hospital, with the complaints of weakness, lassitude, shortness of breath and palpitation.

The patient had apparently been well until nine months before admission, when she suffered from an acute pulmonary condition accompanied by fever, cough and severe prostration. The condition had been diagnosed influenza. She had been acutely ill for only 9 days, but had never regained her strength or felt well afterwards. She had tired very easily and had no appetite or ambition. Within two months of admission she had had an attack of appendicitis. At operation a "slightly inflamed" appendix was removed. She also maintained that the surgeon had noted that there was some peritonitis and that one kidney was small and hard. After operation, she felt so weak that she was compelled to remain in bed most of the time. She suffered from a chronic cough and severe dyspnoea, orthopnoea and cardiac palpitation. Her face and legs gradually became oedematous and she was constantly nauseated and vomited considerably.

In her PAST HISTORY the facts of importance were that she had had pneumonia at the age of 15 months, pertussis at 4 years, and measles at 7 years. She had always been troubled with enuresis.

The FAMILY HISTORY was irrelevant except for the fact that the mother had had two miscarriages.

The PHYSICAL EXAMINATION, on admission, showed severe dyspnoea and orthopnoea, generalized oedema, acrocyanosis, and distention of superficial veins over abdomen and chest. The examination of the lungs revealed dullness at the left base, and flatness at the right base posteriorly. The breath sounds were distant at the left base and almost absent at the right base, posteriorly. Occasional bubbling rales were heard scattered throughout the chest.

The heart was greatly enlarged. The apex was not seen or felt. The dullness to percussion extended 14 cm. to the left and 2.5 cm. to the right of the midsternal line. The aortic dullness measured 6.5 cm. The heart sounds were distant. A distinct gallop rhythm was present. The (P2) second sound in the pulmonary area was not accentuated. A soft systolic murmur was heard at the apex. The murmur was poorly transmitted. The pulse was small and not easily compressed. The peripheral arteries, however, were all thickened and cord-like. The blood pressure was 250 systolic and 190 diastolic. The examina-

tion of the abdomen disclosed a tender, non-pulsating mass, which was considered liver, the border of which was 3 cm. below the right costal margin in the midclavicular line. There was also a moderate amount of fluid in the abdomen.

LABORATORY FINDINGS AND THE RESULTS OF SPECIAL EXAMINATIONS

The urine contained a moderate amount of albumin, and the sediment showed numerous leucocytes and a few hyalin and granular casts. The hemoglobin was 65 per cent, the erythrocyte count 3,200,000, and the leucocyte count 9,800. The non-protein nitrogen was 58 mg. per 100 c. c. of blood. The blood Wassermann reaction was negative. The complement fixation for tuberculosis was negative. The Pirquet skin test was negative.

The electrocardiograms showed low Q. R. S. complexes throughout, diphasic T wave in all leads, and P-R intervals of .160 seconds.

X-ray of the chest showed a homogeneous haziness over the lower portions of both lungs. The more dense shadow was on the right. The heart shadow was greatly enlarged, but the cardiophrenic angle remained acute.

Ophthalmoscopic examination of the eye grounds showed fluffy, slightly elevated discs and the large vessels only about half the normal calibre. Stellate figures were seen at each macula with numerous flame-shaped silvery areas of choroiditis, but no definite hemorrhages.

COURSE IN HOSPITAL: The patient was kept at rest. The fluid intake limited to 1,000 c.c. and a salt free diet was given her. Digitalis and theobromine sodium salicylate were administered with no appreciable effect on her condition. The nausea and vomiting persisted. The blood pressure ranged from 200 to 270 systolic and 180 to 200 diastolic, and was not influenced at all by massive doses of nitro-glycerine. The oedema increased progressively. The cough became worse and the sputum was slightly blood tinged, but never became distinctly purulent, and contained no acid fast bacilli at any examination. The temperature was normal or subnormal during the greater part of the time the child was in the hospital. Once or twice the temperature rose as high as 101 degrees, the remainder of the time it was below 100 degrees. The pulse rate averaged 130 per minute. The non-protein nitrogen of the blood varied from 47 mg. to 58 mg. per 100 c. c.

During the last two weeks of life the patient developed a definite psychosis with hallucinations and delusions. She described many occurrences which had never happened. At times she became violent. Her symptoms were never typical of the classical uremia. There were no symptoms or laboratory signs of acidosis. A few days before death purpuric spots appeared over the abdomen and extremities. Blood cultures taken three days before death were negative. No acute symptoms, convulsions, or coma developed immediately preceding death. The patient had been in the hospital 42 days.

THE CLINICAL DIAGNOSES were: Chronic interstitial nephritis, hypertension, arteriosclerosis, chronic myocarditis, albuminuric retinitis, anasarca, ascites, hydrothorax, and pulmonary atelectasis.

THE PATHOLOGICAL DIAGNOSES were: Chronic diffuse nephritis with small granular kidneys, hypertrophy of the heart, mural thrombi in the auricles, and a ball thrombus in the aorta, infarction of the right lung with an empyema on the right, hemorrhagic infarct of the spleen, fatty degeneration of the liver. There were many subsidiary findings as the general anasarca, ecchymoses into the skin, decubitus ulcers over the sacrum, contractures of the leg muscles, appendectomy scar, pigmentation of the mucosa of the small intestines, prolapsus ani and external hemorrhoids, old pleural adhesions and healed focal calcified tuberculosis of the lung and bronchial lymph glands.

Cultures of the heart blood, pericardial fluid and the purulent exudate from the surface of the diaphragm yielded hemolytic streptococci.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS.

KIDNEYS: The right kidney weighed 103 grams and measured 8.5x5x3.25 cm. The viscus was very firm. The capsule stripped off with ease, tearing the surface at one or two points. The surface was pale and mottled and roughened by projecting nodules of pale yellowish gray tissue with a pinkish gray tissue intervening between the raised areas. A number of opaque yellow spots occurred on the surface. On section the pelvic space was enlarged and filled with fat. The cortex was pale and the few striations that could be made out were seen to be interrupted by opaque spots. The cortex measured 4 mm. Many of the blood vessels cut across in the sectioned did not collapse.

The left kidney weighed 52 grams and measured 7.5x3.5x2.5 cm. It was similar to the right except that it was more contracted and the surface was more granular than that of the right and the small opaque spots were more conspicuous. The cortex measured 3 mm.

THE AORTA was quite elastic. There were many small elevated patches over the inner surface of the arch and about the origins of each of the intercostals. Microscopically, the intima was thickened and showed typical spots of atherosclerosis.

HEART. The pericardium was thickened to 2 mm. and the pericardial cavity contained 20 c. c. of clear yellow fluid.

The heart was displaced slightly to the right and, on severing the aorta during the removal of the heart, a round yellow mass 2.5 in diameter rolled out of the aorta. The mass was tense and light in weight for its size. On section it was composed of an outer capsule of a yellow material resembling fibrous tissue and an inner substance of a deep yellow color, quite opaque and granular in appearance. On removal of the auricles there were seen attached to the walls of the auricles and the appendages (in the left auricle five and in the right auricle one) nodular thrombi similar in makeup to the one described. The mural thrombi varied in size from 3 mm. to 2.5 cm. in diameter.

The ventricular walls were greatly thickened and the papillary muscles were much hypertrophied. The mitral valve was thickened just above its free edge and one segment showed a line of old tough protecting tissue which had a translucent appearance. The other segments also had some thickenings above the free edge and here and there were opaque yellow areas. The free edge and chordae tendinae were delicate. The aortic valve leaflets had quite delicate edges which showed no vegetations. However, the corpora arantii of two of the segments were conspicuous in that they projected 1 mm. above the edge of the valve and measured 1.5 mm. across. The tricuspid and pulmonary valves showed no abnormalities.

Microscopically, the mural thrombi were composed of a deeply staining homogeneous material. At the outer edge the homogeneous material was lamellated to form a capsule for the thrombus. The central portions of the thrombus were quite granular. There were a few red blood cells scattered through the section.

Microscopically the heart valves appeared normal and the subendocardial connective tissue was increased. The subendocardial myocardium showed marked degenerative changes. The muscle cells were slightly swollen and the nuclei were absent. The cytoplasm was very granular. The muscle cells nearest the endocardium were stained a very light pink while those deeper were of a deep rose color, showing a simple necrosis which passed into swelling as one proceeded into the myocardium. There was an increase in connective tissue throughout. In some portions the myocardium showed a marked fatty degenerative infiltration. The subepicardial fat was also increased in amount.

CASE 3—(19-164)

A man, aged 47, was brought into the hospital, complaining of weakness, a pain under the upper sternum, and a sense of constriction, as well as breathlessness, on slight exertion, and also pain in the left shoulder, left chest, and left foot.

The patient attributed all his troubles to an intensive course of intravenous and intramuscular therapy which his physician had given him, following the report of a positive Wassermann reaction in his blood. He had been given 12 intravenous salvarsans and 15 intramuscular mercury injections within a year. At the time of the beginning of his treatment a year before admission he had noticed the high substernal or submanubrial pain, which radiated down over the precordium on exertion. He had also noted a slight but gradually increasing weakness and some loss of weight. The symptoms had increased in severity and the pain became such that it was necessary to raise the left arm above the head and hold the breath to get relief. His sexual power had been lost about four months before admission.

About a month before admission he had noted the onset of severe general weakness which incapacitated him within two weeks. He had suffered a dull pain in the dorsum of the foot, which had been so severe at times that he could not stand on the foot. He had had pain over the left chest, shoulder, and subclavicular regions, and in the left side of the abdomen and the flank. These pains had been rather superficial but severe, and exaggerated by deep breathing. The paroxysms of submanubrial pain accompanied by a sense of constriction and breathlessness became longer and more severe.

In his PAST HISTORY there were practically no facts relevant to his presenting symptoms. He denied having had any venereal diseases. His left eye had been destroyed in an Independence Day accident many years previously.

The MARITAL AND FAMILY histories revealed no important facts.

The PHYSICAL EXAMINATION showed a slightly drowsy male, of a hyposthenic habitus, lying flat in bed apparently not in distress. The skin was sallow and slightly pale. The conjunctivae were pale, but there were no hemorrhages.

The teeth were in very poor condition, many were devitalized. The gums were retracted and pus exuded from the pockets along the alveolar borders.

Strong pulsations were seen in the suprasternal notch and in the carotids. The arch of the aorta was palpable in the suprasternal notch, and a systolic thrill was felt in the carotids.

HEART. The apex impulse was slightly diffuse, but forcible, with the point of maximum intensity in the 5th intercostal space, 9 cm. to the left of the midsternal line.

The border of the cardiac dullness to percussion extended 3 cm. to the right and 10 cm. to the left of midsternal line. The retromanubrial dullness measured 7 cm. There was no other evidence suggesting aneurism. The heart sounds were of about normal intensity. The (A2) second sound in the aortic area was present but faint. The (PP2) second sound in the pulmonary area seemed accentuated. A systolic murmur was heard in the aortic area, whence it was transmitted upward toward the neck vessels. A soft diastolic murmur was also present in the aortic area and transmitted down the left border of the sternum. A short loud systolic murmur was heard in the mitral area and at times transmitted to the axilla. The blood pressure was 140 systolic and 68 diastolic.

There was visible pulsation and throbbing in the thickened tortuous brachials and radials. The radial pulse was quick and suggestive of a Corrigan type. Capillary pulsation was noted. The pistol shot and a Duroziez's sign were heard in the femoral.

In the examination of the lungs, the percussion note was found to be slightly impaired at the right apex, and the breath sounds and vocal fremitus were increased but no rales were found.

The abdomen could not be examined satisfactorily, because of the patient's ticklishness. No tenderness was elicited and the liver and spleen were not palpable.

In the examination of the extremities, clubbed spatulate but non-tender fingertips were noted. The epitrochlear, axillary and inguinal glands were found to be palpable and quite "shotty."

The LABORATORY TESTS AND SPECIAL EXAMINATIONS gave considerable important information. The urine at first showed only a trace of albumin, but later besides the albumin which increased in amount there were found many red blood cells, hyalin and granular casts, and very rare blood cell casts in the urinary sediment. The phenolsulphonephthalein excretion was 55 per cent in two hours on one occasion, and 41 per cent in two hours on another occasion.

The blood presented a polymorphonuclear leucocytosis as high as 20,000 per cubic millimeter as its only abnormality at first. Later a severe secondary anemia developed. The Wassermann reaction in the blood with a cholesterinized antigen was reported two plus on one occasion. The colloidal gold reaction with the patient's spinal fluid yielded a luetic curve. The blood non-protein nitrogen was 35.5 mg. per 100 c. c. The blood calcium on one examination was 9.6 mg. per 100 c. c. Repeated blood cultures showed the *Streptococcus viridans*. Cultures from the apical abscesses showed the same organism.

The X-ray plate of the heart taken at 7 ft. or 2 meters, for the actual cardiac dimensions gave the following figures: M. R. 8.5 cm. M. L. 8.5 cm. L 13.5 cm. A 6.5 cm. The chest was of the long and narrow or asthenic type. The heart shadow and especially the ventricular shadow was transverse. The aorta was long and tortuous with a rather marked prominence to the left, where it turned backward and downward, thus suggesting the arteriosclerotic type of aorta. The electrocardiogram showed no preponderance.

COURSE IN THE HOSPITAL: The patient continued to have an irregular fever throughout his stay in the hospital. The heart murmurs changed slightly, the aortic diastolic murmur became louder, and the mitral systolic murmur became softer.

A large aveolar abscess, about 1.5 cm. in diameter, appeared on the lower jaw. The abscess had originated at the apex of the devitalized right lower molar. The X-ray films showed two other apical abscesses. The left foot and ankle flared up, at the same time, became oedematous, hot, and red.

The spleen became palpable and pain and tenderness were noted in the splenic region. Nosebleeds and bleeding from the alveolar cavities from which teeth had been extracted were troublesome.

Crops of petechiae appeared over both legs below the knees. The lesions were irregularly scattered 2 to 10 mm. in diameter with a tendency to grow larger. The blood pressure gradually dropped to 100 systolic and 40 diastolic.

The massive oedema of the legs gradually increased.

The sternum became tender and the substernal pain required large doses of morphine to allay it. The patient suffered sudden attacks of headache and disorientation.

He gradually grew worse and died 102 days after admission.

The **CLINICAL DIAGNOSES** were: Subacute bacterial endocarditis, (streptococcus viridans endocarditis), chronic cardiac valvular disease, aortic regurgitation, aortitis, syphilis, multiple infarcts and abscesses, hemorrhagic nephritis, general anasarca, splenic enlargement, suppurative ethmoidal and sphenoidal sinusitis, chronic tonsillitis, and apical abscesses.

The **PATHOLOGICAL DIAGNOSES** were: Acute and chronic endocarditis, mural thrombi, pericarditis, beginning aneurism of the root of the aorta, aortitis, acute and chronic nephritis, acute splenic tumor with infarcts, hydrothorax, bilateral oedema of the extremities, multiple ulceration of the forehead and hands. Bilateral healed apical tuberculosis, fibrous adhesive basal pleuritis, fibrous peritonitis in the gall bladder and appendix regions, and fibrous perisplenitis.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS.

KIDNEYS: The right kidney weighed 200 grams and measured 13x8x3 cm. The left weighed 210 grams and measured 13x8.5x3.5 cm. The capsules stripped with ease and left smooth surfaces of a deep purplish red color. The same color was seen throughout the organ on section. There was a grayish cast to the color on the cut surface and the normal markings were obliterated.

Microscopically, the evidences of an extensive congestion were seen. The tubular cells were swollen and granular and in places disintegrated. Many tubules contained casts. There were areas of scarring with mononuclear and polynuclear cell infiltration.

The **AORTA** was fairly elastic and contained many small atheromatous areas. The root had many fatty plaques, some of which showed calcification. One area, 1.5 cm. in diameter, was eroded. Microscopically, there was much infiltration of the intima with small mononuclear cells, and the vasovasorum were thickened.

HEART: The heart weighed 420 grams. Scattered over the pericardium along the course of the vessels were numerous small atheromatous areas. The valves measured as follows: Tricuspid 11 cm., pulmonary 7 cm., mitral 10 cm., and aortic 7.5 cm. The tricuspid and pulmonary valves were free from vegetations. The mitral and aortic valves had numerous vegetations on all the leaflets. The largest of the vegetations was on the posterior mitral leaflets and measured 3 mm. in diameter. Slight extension down the chordae tendinae was noted. Behind the right posterior aortic leaflet there was a mycotic aneurism 6 mm. in diameter bulging into the anterior mitral flap. The aortic ring contained many small areas of calcification. The myocardium was brown in color.

Microscopically, the valves were thickened and hyalinized and showed a diffuse infiltration with polymorphonuclear cells. Attached to the valve at one point was a mass of fibrin showing lamination; at its base there was beginning organization with ovoid and globular nuclei and few polymorphonuclear cells.

Microscopically, there were evidences of a very marked chronic myocarditis. The subepicardial fat was increased in amount. There was a mononuclear infiltration of the subepicardial fat. The muscle fibers showed various stages of degeneration, some simple necrosis, some fatty degenerative infiltration, and some cloudy swelling. There was a marked fibroblastic and angioblastic proliferation. Many of the cells contained a light yellow diffuse pigment. There was some endocardial thickening.

CASE 4—(48-78)

A man, age 35, was admitted to the hospital with the complaints of shortness of breath, palpitation, headache, sleeplessness, and cough.

The patient had been aware of the presence of albumin in the urine since an attack of acute nephritis with oedema, which had followed a series of winter colds and tonsillitis, twenty years previous to admission. A distinct elevation of the blood pressure was known to have been present for at least two years. From this time on his protein intake had been moderately restricted. The phenolsulphonethalein excretion had been reduced to 45 per cent, one and one half years before admission. Six months before admission an ophthalmologist had found an extensive retinal hemorrhage. The systolic blood pressure at this time had been found to be 200. At this time he had begun to sharply curtail his very active life. A month before admission the blood pressure had been found to be "over 200," and shortness of breath had been noticed.

The **ACUTE ILLNESS**, however, had begun seventeen days before admission as a respir-

atory infection with a chill, cough, expectoration, slight fever, headache, indigestion, a tender spot in the epigastrium, and gaseous distention. Three days after the chill, he had an acute attack of dyspnoea, which was relieved by morphine and digitalis. He became greatly depressed mentally and developed insomnia. A soreness in the eyes, headache, and a cough persisted.

On PHYSICAL EXAMINATION, the patient was found to be quite drowsy. The breath was heavy and had a urinous odor. Ophthalmoscopic examination showed many retinal hemorrhages in the right fundus. The retinal vessels were moderately sclerosed.

The heart was enlarged. The apex was seen and felt as a forceful impulse, accompanied by a systolic shock and thrill. The point of maximum intensity of the apex impact was 11 cm. to the left of the midsternal line. The first sound at the apex was loud and booming. The aortic second sound was very much accentuated and had a metallic ringing quality to it. The cardiac rhythm was disturbed by occasional extrasystoles. No murmurs were heard. The blood pressure was 228 systolic and 148 diastolic.

The abdomen was slightly distended but not rigid or tender to touch. The liver edge was not palpable.

There was no oedema present.

The RESULTS OF LABORATORY TESTS AND OTHER SPECIAL EXAMINATIONS were significant.

The urine output was good on admission, but fell off sharply in volume in spite of a sustained intake. The specific gravity was never above 1.014. Four grams of albumin were present in each liter. The sediment showed a few hyalin and many short dark granular casts; later cellular and fatty casts, and fat globules were present. The phtalein output was but 5 per cent in two hours on admission, later none of the dye was excreted in two hours. The blood showed a moderately severe secondary anemia. The quantitative blood chemical analyses showed a distinct and progressive retention of nitrogenous bodies, the non-protein nitrogen had been 102 mg. per 100 c.c. just before his admission to the hospital, but it gradually rose to 172.5 mg. The uric acid fraction had been 6.82 mg. at first and rose to 7.12 mg. the creatin had been 4.68 and rose to 5.19 mg. per 100 c.c. The Wassermann reaction was negative.

The dimension measurements on the seven foot heart plate were M. R. 6 M. L. 9.5 L. 18 A.6. cm.

The electrocardiograms showed a digitalis effect on the T wave, but no signs of preponderance were present.

COURSE IN THE HOSPITAL: The patient became progressively worse in spite of all dietetic restriction, elimination procedures, and cardiac supportive treatment. Five hundred c. c. of blood were removed and an equal volume of 5 per cent glucose given by vein puncture, without avail.

During the third night in the hospital, the patient had an attack of severe epigastric pain and vomiting. These symptoms persisted.

A complicating abdominal distention became very severe and failed to respond to any therapy. The patient became distinctly uremic with facial twitchings, delirium and restlessness.

The heart outline increased, an apical systolic murmur, that was transmitted over the precordium and to the axilla, developed. The heart rate rose to 140 per minute and a temperature of 100 was recorded. The breathing was of the Cheyne-Stokes type, the dyspnoea increased and rhonchi were heard throughout the right lung. The patient died on the eighth day after admission.

The CLINICAL DIAGNOSES were: Chronic interstitial nephritis with a superimposed acute parenchymatous nephritis, acute uremia, chronic myocarditis, cardiac hypertrophy and dilatation, hypertension, arteriosclerosis general, albuminuric retinitis with hemorrhages.

The PATHOLOGICAL DIAGNOSES were: Chronic diffuse nephritis, coronary and peripheral arteriosclerosis, hypertrophy of the heart, focal epicarditis with hemorrhages, bilateral hypertrophy of the adrenal cortex, focal gangrenous enteritis of the jejunum, acute splenic tumor, hyperplasia of the malpighian corpuscles, fibrino-purulent peritonitis chronic passive congestion of the lungs, congenital obliteration of the gall bladder. The blood cultures and cultures of the peritoneal exudate yielded hemolytic streptococci.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS.

KIDNEYS: The kidneys were small, weighing only 80 grams each measuring 9x5x2.5 cm. The capsules stripped easily exposing a mottled granular surface, due to the intermingling of deep red and yellowish foci. The cut surfaces were similarly mottled. The normal kidney markings were obliterated, irregular lines and grayish irregularly outlined foci were scattered in the cortical regions of both kidneys. The normal lines of cortical demarcation could not be made out.

Microscopically, many of the tubules, especially the convoluted ones, were filled with hyalin casts. There was an advanced degeneration of the tubular epithelium. Many large areas showed a heavy round cell infiltration and connective tissue proliferation. The arcuate arteries were very sclerotic. The glomerular changes were variable, some were converted into scars, others were hyalinized, while in some the capillary tuft was shrunken and the space within the capsule taken up by granular material.

The AORTA was on the whole remarkably free from sclerosis and consequently quite elastic. This was especially so in the thoracic and abdominal portions, while the arch showed a few small atheromatous foci just above the aortic valves. Microscopically there was a slight thickening of the intima and connective tissue proliferation in the media.

HEART. The pericardial fluid was slightly increased and turbid. The epicardium was finely granular in places. Petechial hemorrhages were quite numerous at the auriculo-ventricular junction.

Both coronary arteries were markedly sclerotic. The process was especially advanced in the right coronary artery.

The valves measured as follows: Aortic 9.5 cm., mitral 12.5 cm., pulmonary 9 cm., tricuspid 16 cm. The valves all appeared normal.

Microscopically, there was a slight fibrosis throughout the sections. The muscle cells seemed slightly smaller than normal and most of them showed a fair degree of cloudy swelling. In various places throughout the sections there was more or less diffuse round

cell infiltration into the myocardium. The epicardium and endocardium were only slightly thickened. The majority of the blood vessels showed a marked thickening. In the subepicardial fat along one side there was a very marked infiltration of mononuclear cells, the majority of which were plasma cells. In this same area there were both old and recent petechial hemorrhages. The mononuclear infiltration extended into the myocardium.

CASE 5—(3-3)

A man, age 32, was brought to the hospital because of severe nose bleeds, weakness, failing vision, headaches, shortness of breath, and pain in the chest.

The symptoms which caused him to seek medical aid were all dated from a severe attack of "Influenza" ten months previous to his admission. He had been told by his doctor that he had not had a pneumonia, nevertheless, he had been very sick with a high fever which had kept him in bed for three weeks. Previous to this, he had had occasional headaches, but had noted no change in vision. He maintained that he had felt "fairly good" after his recovery from the "influenza" and had been able to do hard farm work and endure considerable exposure. He had begun, however, to suffer from severe headaches and failing of vision. On one occasion six months before admission, he had returned from work at noon, on account of a very severe throbbing headache, had seated himself and then had fallen to the floor unconscious. The unconscious state or stupor was said to have lasted for three days, and to have been followed by an excitement stage, in which the patient had to be held in bed. Within a week, however, he was working hard again and consuming large amounts of all kinds of foods. The severe headache persisted, but in spite of it and frequent attacks of unconsciousness he had worked regularly all summer. He had fallen unconscious following a five mile walk four months previously. Shortness of breath, oedema of the dependent parts, morning nausea and vomiting, and insomnia were noted at about this time. The symptoms were relieved and the oedema disappeared with rest in bed for two weeks.

Three weeks before admission his first attack of epistaxis began following a ten mile ride on horseback. The bleeding continued for 14 hours, and was accompanied by profuse sweating and was followed by extreme weakness. He continued to have nosebleeds daily, and as a result his headache seemed less severe, and his vision seemed to improve. The vomiting continued. A slight hacking cough, fever and pain in the chest developed in the week just previous to his admission.

The PAST HISTORY was negative except for the patient's having had the ordinary diseases of childhood without complications and an uncomplicated typhoid fever at the age of 13.

The FAMILY HISTORY and MARITAL HISTORY were entirely negative.

The PHYSICAL EXAMINATION showed the powerful build of the patient. The facies were, however, pale, sallow, waxy, and puffed. The patient was drowsy and his sensorium was clouded. The mucous membranes were pale, the gums were swollen and dirty with much pus along the alveolar border. Two subconjunctival hemorrhages were present just outside the left cornea.

The neck veins were full and showed a systolic pulsation. There was much throbbing above the right clavicle.

HEART. The cardiac apex was seen and felt in the 6th intercostal space, the point of maximum intensity was 12.5 cm. to the left of the midsternal line. There was a sharp systolic shock, a faint impulse and a diastolic shock just inside and below the nipple. No definite friction could be felt. The dullness to percussion extended 5 cm. to the right and 13 cm. to the left of the midsternal line. The aortic dullness measured 6 cm. The rhythm was that of an embryocardia. The pulmonary second sound was accentuated. The aortic second sound was loud and of a liquid quality. The heart sounds were well transmitted. A to-and-fro leathery friction rub was heard over the precordium and in the left axilla. The blood pressure was 160 systolic and 130 diastolic. The pulse was small, firm, and regular at the rate of 125 per minute. Alternation was noted at times. The peripheral vessel walls were markedly thickened and tortuous. There were signs of fluid at the left base.

The liver border was felt 6 cm. below the right costal margin in the midclavicular line.

There was considerable oedema noted in the pretibial tissues.

The LABORATORY TESTS AND SPECIAL EXAMINATIONS yielded many significant facts. The urine output was low and the specific gravity was never above 1.013. It was found to contain 3 to 5 grams of albumin per liter, and the sediment showed many waxy, granular and hyalin casts. No 'phthalein was excreted in two hours.

The blood showed a very severe secondary anemia with red blood cell count always between 1,300,000 and 1,800,000 and a hemoglobin percent of 20 to 24. The leucocyte counts were between 9,000 and 10,000. The blood non-protein nitrogen was 167 mg. before and 250 mg. per 100 c. c. after transfusion. Blood cultures were negative. The Wassermann was negative.

An X-ray plate taken at 7 feet gave the following measurements: M. R. 6.5 cm., M. L. 14.5, L. 20.5, and A. 8.5 cm. A diagnosis of pericarditis was also made.

Ophthalmoscope examination revealed oedema of the retina and the disc, with much exudate. There were no fresh hemorrhages.

Pulsus alternans was frequently noted and tracings were made of the disturbance.

The electrocardiograms showed slight left ventricular preponderance and a slightly lengthened P-R interval.

COURSE IN THE HOSPITAL: Catheterization was necessary every eight hours. The patient's nose bled frequently. A transfusion of 500 c. c. of blood was of no great help. A clear yellow exudate was removed from the left pleural cavity in 700 and 900 c. c. amounts on two occasions. The pericardial friction rub became louder and then almost disappeared.

A terminal bronchopneumonia developed at the left base and the patient died 10 days after admission.

The CLINICAL DIAGNOSES were: Chronic interstitial nephritis with acute parenchymatous degeneration, chronic uraemia, acute pericarditis, hypertension, cardiac hypertrophy and dilatation, chronic myocarditis, left-sided serofibrinous pleurisy, epistaxis, left

spermatocele and chronic epididymitis and severe secondary anemia.

The PATHOLOGICAL DIAGNOSES were: Chronic diffuse nephritis with widespread degeneration, fibrinopurulent pericarditis, cardiac hypertrophy and dilatation, left acute fibrinopurulent pleurisy, acute cystitis, chronic prostatitis and epididymitis, and left spermatocele.

GROSS AND MICROSCOPICAL DESCRIPTIONS

KIDNEYS: The kidneys weighed 225 grams each and measured 14x6.5x3 cm. each. The capsules stripped with ease, leaving a pale grayish white oedematous surface. The cortical striations were lost in the diffuse grayish white tissue with the yellow opaque areas scattered here and there.

Microscopically, the kidney sections presented a diffuse degenerative nephritis. The few intact cells that were scattered here and there showed cloudy swelling. The tubules contained much granular debris and many granular casts. Some areas of fibrous tissue with round cell infiltration were noted. Some glomeruli were completely necrosed, others contained varying amounts of granular debris and showed some fibrous thickening of the capsule.

The AORTA was moderately sclerosed with raised yellow plaques throughout. The elasticity was reduced. Microscopically, there were atheromatous areas and intimal thickening.

The HEART was considerably enlarged. The pericardium was distended with about 150 c. c. of fibrinopurulent exudate. The visceral pericardium was covered with shaggy fibrin masses.

The valves were slightly thickened but were otherwise negative. A thin antemortem clot was found fixed in the trabeculations of the right ventricle.

Microscopically, the majority of the muscle cells showed a slight cloudy swelling. The blood vessel walls were very much thickened throughout all the sections. The epicardium was very much thickened and in some places was completely lost or at least replaced by a leucocytic infiltration with many irregular masses of blue staining granular bodies, which were colonies of bacteria. In this and other regions in the subepicardial connective tissue and in the fat, there was a marked angioblastic and fibroblastic proliferation, indicating a chronic condition. There was also many large and small foci of polymorphonuclear leucocytes, in the myocardium signifying a myocarditis. This process was also chronic as shown by the angioblastic and fibroblastic proliferation. Along with the colonies of bacteria there were areas of simple and coagulation necrosis around which there were areas of advanced cloudy swelling.

CASE 6—(47-79)

A negro, age 38, was admitted to the hospital, complaining of severe headache, vomiting and a loss of weight.

His illness had had an insidious onset. Headache, dizziness, nocturia and frequency had been present for three years, and had been rather rapidly increasing in severity for two years. Protein foods, especially meats, had been restricted for three years, upon the advice of a physician. His blood pressure had been 210 mm. mercury systolic for at least two years. The progress of his trouble had been unaffected by treatment. All the above mentioned symptoms had become more severe, and in addition he had been troubled with severe vomiting and blurring of vision. He had lost 35 pounds of weight. He had had no oedema.

In his PAST HISTORY he had had frequent attacks of tonsillitis in youth. He had had gonorrhea at the ages of 19, 25, and 31 years. With the last attack he had frequency, dysuria, hematuria, pyuria, and pyrexia, which had cleared up after the dilatation of the urinary sphincter and irrigation of the bladder. He had also a pneumonia at 31 and dysentery at 32.

The PHYSICAL EXAMINATION showed carious teeth and severe pyorrhea. Pus was expressed from the gums and the tonsils.

The heart was enlarged. The apex impulse was diffuse and heaving. The retromammary dullness was increased. The (A2) second sound in the aortic area was accentuated and ringing. The (M1) first sound in the mitral area was loud and booming and accompanied by a slight systolic murmur. The blood pressure was 265 mm. mercury systolic and 145, diastolic. The brachial and radial arteries were thickened and tortuous.

The liver edge was just palpable, but was not tender. The prostate was enlarged and boggy.

The significant LABORATORY FINDINGS were as follows: The urinary output was slightly increased in amount, with the night volume usually greater than the day volume. The specific gravity was fixed at 1010 and the chloride output was constantly low. Two grams of albumin were present in each liter of urine. Hyalin and coarsely granular casts were present in small numbers. At the time of admission the 'phthalein output was 25 per cent in two hours. The blood showed a hemoglobin of 75 per cent, a red count of 3,980,000, a white count of 8,800 and a normal differential count. The blood Wassermann was negative. The blood non-protein nitrogen was 55.7 mg., creatinin 4.54 mg. and uric acid 6.26 mg. per 100 c. c.

The ophthalmologist found small scattered areas of retinitis and retinal hemorrhages. The genito-urinary consultant diagnosed a chronic prostatitis.

A heart X-ray plate gave the following dimensions: M. R. 5.5 cm., M. L. 9.5 cm., L. 18.5 cm., A. 6.5 cm.

X-ray plates of the arteries of the leg demonstrated calcification of the posterior tibial.

Electrocardiograms showed left ventricular preponderance.

THE COURSE IN THE HOSPITAL was one of moderately rapid downward progression in spite of the treatment. A broncho-pneumonia developed after he had been in the hospital about three weeks. The concentration diuresis test indicated almost complete kidney insufficiency. The 'phthalein output was 8 per cent, the blood creatinin was 4.41 mg., and the uric acid 5.27 mg. per 100 c. c. The bronchopneumonia, which the sputum culture showed to be due to a group IV pneumococcus and a hemolytic streptococcus, proved fatal in about five days.

The CLINICAL DIAGNOSES were: bilateral bronchopneumonia, chronic interstitial nephritis, chronic myocarditis, hypertension, albuminuric retinitis, general arteriosclerosis, chronic prostatitis, dermatitis seborrhoeica, chronic tonsillitis, dental caries, pyorrhea alveolaris, and chronic constipation.

The PATHOLOGICAL DIAGNOSES were: chronic diffuse nephritis, with arteriosclerotic kidneys, moderate arteriosclerosis, cardiac hypertrophy and dilatation, acute aortic vegetative endocarditis, chronic myocarditis, chronic passive congestion of spleen and lungs, sub-acute diphtheritic, cystitis, chronic prostatitis, and bilateral bronchopneumonia.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS.

KIDNEYS: The right weighed 180 grams, the left 100 grams; the right measured 12x7x3 cm., the left 12x6x3 cm. The pelvis showed numerous injected areas. The capsules were slightly adherent and the surfaces were granular. There were numerous little yellow areas throughout, intermingled with gray yellow and gray opaque areas. Numerous injected spots could be seen on the surface. A few cortical cysts were present. On section, the striations of the cortex were not clearly defined. The renal vessels showed mild sclerosis. The iodine tests for amyloid were negative.

Microscopically, the kidneys showed a chronic diffuse nephritis. The tubules showed varying degrees of degeneration. Many contained large hyalin casts, while some contained polymorphonuclear leucocytes. The glomeruli were the seat of chronic changes, with proliferation of the capsular epithelium in some and fibrotic changes in others, varying from a slight amount to a complete fibrosis. The interstitial tissue was increased in amount and there was lymphocytic accumulation in many places. The blood vessels showed a striking amount of sclerosis, which in some places almost completely obliterated the lumen.

The AORTA showed moderate sclerosis and was blood stained.

Microscopically, there was a distinct thickening of the intima with atheromatous patches scattered here and there.

The pericardium was smooth, moist and glistening throughout.

The HEART weighed 615 G. The auricles and ventricles showed definite dilatation and hypertrophy. The ventricles alone weighed 495 grams. The valves measured as follows: tricuspid 13 cm., mitral 10.5 cm., pulmonary 7 cm., and aortic 6.5 cm. The aortic leaflets showed a string of minute vegetations at the edge of each cusp. The tricuspid valve leaflets showed red areas that suggested oedema of the valve.

Microscopically, the aortic valve cusps showed well organized vegetation. The endocardium was markedly thickened and in one section showed a mixed clot attached to it. The endocardium also presented a marked leucocytic infiltration. The clot contained many polymorphonuclear leucocytes. The subendocardial connective tissue was increased in amount and showed a marked infiltration of leucocytes also. The myocardium showed a marked fibrosis and a very large number of diffusely scattered leucocytes. There was also some angioblastic proliferation indicating chronicity. Some of the muscle fibers showed simple necrosis while others showed only cloudy swelling. The subepicardial connective tissue was increased in amount and was densely infiltrated with polymorphonuclear leucocytes. The fibroblastic and angioblastic proliferation indicated chronicity of this lesion also. There was a chronic passive congestion of the blood vessels. The blood vessels were mostly sclerotic.

CASE 7—(37-89)

A widow, aged 60 years, was admitted to the hospital, with the complaint of jaundice together with pain and swelling of the abdomen.

Her PRESENT ILLNESS was dated from an acute attack of abdominal pain one year before admission. The pain was sharp and was located under the right costal margin and radiated to the right back only. It lasted two weeks and was accompanied by nausea and was followed by jaundice, dark colored urine and clay colored stools. After a few months the abdomen became enlarged. Oedema of the feet and legs had been present for two months. There had been a loss of 75 pounds in weight within a year.

The past history, family history and marital history were unimportant.

The PHYSICAL EXAMINATION showed an apathetic old woman with emaciation and cachexia, a chocolate brown pigmentation of the skin and a deep almost black jaundice of the sclerae and mucus membranes. The cervical glands were enlarged.

The lungs showed dullness at both apices and the bases posteriorly. The breath sounds and whispered voice were increased and rales were present at the bases.

The heart apex impulse was seen and felt in the 5th intercostal space 8.5 cm. to the left of the midsternal line. The heart sounds were distant and no murmurs were heard. The blood pressure was 85 mm. mercury systolic and 40 diastolic.

The abdomen was bulging in the flanks and there was shifting dullness and a fluid wave present. The veins over the abdomen were enlarged. The liver edge was just palpable.

The LABORATORY EXAMINATIONS revealed the following: The urine showed bile but was otherwise negative. No Bence-Jones protein was present.

The blood showed 60 per cent hemoglobin, 3,000,000 red blood cells, 7,250 white blood cells; a blood calcium content of 8.9 mg. per 100 c. c. and a negative fibrinolysin test and Wassermann test. The stools showed bile pigment and a positive benzidine reaction. She frequently vomited coffee ground material and occasionally fresh blood.

The gastric analysis showed a normal acidity curve. The gastro-intestinal fluoroscopic examination revealed nothing of significance. The sternum was fractured by light pressure. The X-ray plate of the chest was interpreted as showing evidence of pulmonary tuberculosis, dilation of the aortic arch, arteriosclerosis and bone atrophy. The sputum was repeatedly negative.

The ascitic fluid was greenish yellow with a specific gravity of 1008 to 1012, and contained 5 gms. of albumin per liter, a moderate number of small mononuclear cells, a few polynuclears and a few large endothelial cells. Exploratory laparotomy showed an atrophic, very dark green slightly nodular liver.

The electrocardiograms showed very small complexes and a slight left ventricular preponderance.

She gradually lost ground and died, two and one-half months after admission.

The CLINICAL DIAGNOSES were: Carcinoma of the liver, cirrhosis of the liver, oesophageal varices, arteriosclerosis general, chronic bronchitis, healed apical tuberculosis, osteoporosis, fracture of the sternum, peritonitis chronic ascites, icterus, cholelithiasis, adrenal insufficiency.

The PATHOLOGICAL DIAGNOSES were: Carcinoma of the hepatic duct with obstruction and invasion of the liver, cirrhosis of the liver, oesophageal varices, ascites, jaundice, hypostatic pneumonia, general arteriosclerosis and chronic nephritis with cysts. There were many subsidiary pathological processes such as bilateral fibrous pleurisy, tuberculous peribronchial lymphadenitis, osteoporosis with the fracture of three ribs and the lower sternum, chronic pancreatitis, calcified tubercles of the spleen and accessory spleen and a perisplenitis.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS

KIDNEYS: The right kidney weighed 100 grams and measured 9.5x5x3 cm. The capsule was definitely thickened and the kidney tissue was adherent. Small retention cysts 3 mm. and 8 mm. in diameter were present in the upper pole. On section the kidney was firm and of an opaque yellow brown color. The left kidney weighed 110 grams and measured 10x5x3 cm. The appearance was quite similar to that of the right, besides small cysts, however, gray white nodules were present on the surface. The pelvic fat appeared gelatinous. No sclerosis of the vessels was present.

Microscopically the kidneys showed many bile stained and hyalin casts. The glomeruli were very sclerotic to the point of being partially replaced by fibrosis. The tubules contained much granular sediment. The arterioles were markedly sclerosed.

The AORTA showed considerable arteriosclerosis with numerous hard bile stained plaques especially about the branches. The intima was distinctly thickened.

The HEART showed moderate coronary sclerosis. The valves presented no gross abnormalities but microscopically there was an intimal sclerosis.

The muscle cells were slightly smaller than normal and some of them showed a slight cloudy swelling. There was a slight increase in the subepicardial connective tissue. The muscle and endocardium were not abnormal.

CASE 8—(1-1)

A widow, aged 78 years, was brought into the hospital on a stretcher complaining of pain under the lower sternum, shortness of breath, weakness, loss of appetite and a decrease in the output of urine.

Her PRESENT ILLNESS was dated from a fall eight years previous to her admission. The onset of her failing health was quite insidious, with weakness as her only symptom. Later she noted a numbness and coldness of her feet without any loss of the use of the extremities. However she was in bed for two years. Then she got up again, but soon noted an increasing weakness and had to give up, and for five years she had been more or less bedridden. Along with the great loss of strength she had lost some weight and had suffered from a loss of appetite and nausea. She had had some dyspnoea, palpitation, paraesthesia and coldness of the extremities, and some sputum but no cough.

The PAST HISTORY was not significant except for exposure to tuberculosis from which her father, brother and sister had died. The family history was otherwise negative.

The PHYSICAL EXAMINATION revealed a tall, cachectic, haggard, old woman, who seemed listless and mentally confused. The musculature was completely atrophied. The skin had lost all elasticity and hung loosely in folds and contained no panniculus.

The chest was hyposthenic and rachitic with a deep groove and flaring costal margins. The heart seemed to be displaced to the left. The point of maximum intensity of the fairly diffuse apex impulse was in the 5th intercostal space 12 cm. to the left of the midsternal line. The left heart border dullness corresponded, the rest of the cardiac out line could not be determined because of the emphysematous tympany. The heart sounds were fairly loud and rapid, averaging about 120 per minute, and the rhythm was quite regular except for extrasystoles. A soft blowing systolic murmur was heard at the apex. The blood pressure was 170 mm. mercury systolic and 100 diastolic. The brachials and radials were sclerotic thickened and beaded.

The lungs were emphysematous. Dullness was present over the right back from the spine to the angle of the scapula and for 8 cm. out from the spinous processes. Over this area the breath sounds were diminished while further toward the axilla they were amphoric. The breath sounds otherwise were bronchovesicular. Rales were heard at the bases.

The abdomen was scaphoid and a distasis of the recti was present. The liver was hard, irregularly nodular and slightly tender and extended 11 cm. below the costal margin.

The laboratory examinations revealed some disturbance in kidney function and a secondary anemia. The urine was small in amount and of moderately low specific gravity and contained albumin casts and leucocytes. The 'phthalein output was 32 per cent in two hours. The blood showed a hemoglobin of 50 per cent, 3,450,000 red blood cells and 9,200 white blood cells with a normal differential count. The non-protein nitrogen was 49 mm. per 100 c. c. and the Wassermann was negative.

The sputum was negative.

Ophthalmoscopic examination showed "crinkly" retinal vessels.

Electrocardiograms revealed auricular flutter and left ventricular preponderance. Digitalis changed the rhythm to auricular fibrillation and then to normal mechanism with auricular extrasystoles.

Her condition seemed unchanged until her death which came suddenly, on the thirteenth day after admission.

The CLINICAL DIAGNOSES were: Carcinoma of the liver, general arteriosclerosis, chronic myocarditis, auricular flutter and fibrillation, normal mechanism with auricular ex-

trastystoles, cardiac hypertrophy and displacement, hypertension, chronic nephritis, and chronic pulmonary tuberculosis.

The PATHOLOGICAL DIAGNOSES were: Squamous cell carcinoma of the bladder, with metastases to the retroperitoneal lymph nodes, liver and pleura. Occlusion of the left ureter, left hydronephrosis, chronic aortitis with aneurism of the ascending aorta, general arteriosclerosis, arcus senilis, cardiac hypertrophy, chronic passive congestion of the viscera, anasarca and emaciation.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS.

KIDNEYS: The left kidney weighed 155 grams and measured 12.5x5.5x3 cm. while the right weighed 125 grams and measured 11x5.5x3 cm. The pelvis of the left kidney was distended with urine and the left ureter was dilated to the size of a finger. The capsules stripped with ease. On section the kidney substance was pale and undifferentiated.

Microscopically there was a chronic diffuse nephritis with scarring. The vessels were sclerotic.

The HEART was displaced far to the left by a large dilatation of the ascending aorta which was both sacculated and fusiform, extending from the root of the aorta to the origin of the innominate artery, a laminated clot filled the sacculated aneurysm.

The AORTA was extensively sclerosed throughout. The abdominal portion showed hard scales or plates protruding into the lumen.

Microscopically, the aorta below the aneurysm showed a fibrous thickening of the intima with degeneration and vacuolization with focal areas of hyalinization replacing much of the intima and media. At the edge of the aneurysm the fibrous media passed into and became continuous with a thick mass of coarse hyalinized tissue, along the outer edge of which were clusters of small mononuclear cells in perivascular arrangement. These same cellular collections were seen in the intima.

The HEART itself was not very large but the left ventricle appeared hypertrophied.

Microscopically, there was a well marked fibrosis present which was more prominent in some places than in others. The muscle cells seemed to be much smaller than normal. In some places there were calcium deposits in the subendocardial connective tissue. The blood vessel walls were markedly thickened. In the muscle fibers there were cones of yellowish brown pigment extending from the poles of the nuclei. This was hemofuscin pigment and indicated brown atrophy of the heart. The epicardium showed no abnormalities other than a slight increase in subepicardial fat.

CASE 9—(33-93)

A widow, aged 60, entered the hospital with the complaint of pain in the right lumbar region and flank.

Her PRESENT ILLNESS was attributed to the drinking of a large quantity of possibly contaminated water eight weeks previously. Within a few days after the supposed infection she felt dizzy and nauseated and had a dull pain in the right lumbar region. The pain was sharp and more severe at night often requiring morphine. There was relief during the day, except for a slight ache. The pain gradually extended around to the right lower quadrant. The right hip became painful and the right leg could not be raised. Chills, fever and sweats were present for a few weeks. She had lost considerable weight and strength and had been on a diet for the first two years after the diagnosis had been made. Her menses on some days and much less on others, but a nocturia of two to three times had been constantly troublesome.

Her PAST HISTORY was significant in that she had had diabetes mellitus for ten years and had been on a diet for the first two years after the diagnosis had been made. Her marital history was irrelevant. The family history was of some importance since one sister had died of diabetes.

The PHYSICAL EXAMINATION showed a somewhat pale but moderately obese woman. The teeth had all been removed.

The chest was rather narrow in the region of the shoulder girdles. The lungs were negative.

The heart's apex impulse was not seen but the point of maximum intensity was palpated in the 5th intercostal space 11 cm. from the midsternal line. The cardiac pulsations were felt all over the precordium. A systolic shock accompanied the strong apex heave. The heart was slightly enlarged. The heart sounds were clear and the rhythm was regular except for occasional extrasystoles. The aortic second sound was slightly ringing. The first sound at the apex was somewhat impure while the second was clear and loud. A soft blowing systolic murmur was heard at the apex. The blood pressure ranged from 160 to 180 mm. mercury systolic and from 80 to 100 diastolic. The radial arteries were slightly thickened and tortuous.

The abdomen was large. A tender, indefinitely outlined, indurated, flat, nodular mass filled the whole right flank. The superior border was most easily made out. There was no movement with respiration.

The LABORATORY EXAMINATION gave much useful information. The urine contained 2 grams of albumin and 26 grams of sugar per liter. There were very many coarse and finely granular casts and a few leucocytes in the sediment. The phthalein output mounted to only about 10 per cent in two hours.

The blood showed 56 per cent hemoglobin, 4,100,000 red blood cells, and 12 to 20,000 leucocytes with a preponderance of the polymorphonuclears in the differential count. The blood sugar was .44 per cent and .26 per cent; the CO₂ 59.7 Vol. per cent. The Wassermann was negative.

The X-ray plates showed renal calculi on the right side. A seven foot heart plate gave the following dimensions: MR. 45, ML. 95, L. 16, and A 5 cm.

The electrocardiogram showed a left ventricular preponderance.

COURSE IN HOSPITAL: A right nephrostomy was done, and 300 to 400 c. c. of greenish yellow odorless pus was drained off. The temperature fell to normal and the kidney abscess continued to drain well. While convalescence was progressing she suddenly

felt faint and collapsed, becoming pulseless and cyanotic, she gasped a few times and died. The CLINICAL DIAGNOSES were: Cerebral embolism, diabetes mellitus, right pyonephrosis and nephrolithiasis, chronic nephritis, arteriosclerosis.

The PATHOLOGICAL DIAGNOSES were: Right nephrolithiasis and pyonephrosis, nephrotomy with drainage, acute ureteritis on the right with obstruction at about the middle third of the ureter, chronic cystitis, hypertrophy of left kidney, fibrinous perisplenitis and perihepatitis, acute aortic vegetative endocarditis, atrophy and lipomatosis of the pancreas, hyperplasia of the malpighian corpuscles, moderate sclerosis of the aorta and the coronary arteries.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS

KIDNEYS: The left kidney weighed 255 gms. and measured 15x7x4 cm. It was congested and showed an acute diffuse nephritis. The right kidney was bound down by adhesions. Numerous abscesses were present throughout the kidney substance which had been almost entirely replaced by fat and fibrous tissue. The calyces were distended with pus. The operative opening drained one of the calyces. Two small stones were present in the calyces and one large stone filled and almost entirely blocked the pelvis. The right ureter was large and thick with an obstruction at about its middle. The ureter was reddened and thickened.

Microscopically, the right kidney showed almost complete disappearance of kidney substance; only a few tubules and no glomeruli were present. There was considerable oedema, hemorrhage and infiltrations with polymorphonuclear leucocytes, with fibrin in the mass of fibrous tissue and fat. The left kidney showed lymphocytic accumulations in places with occasional leucocytes in the glomerular capsule and some fibrosis. The picture was that of a chronic diffuse nephritis with very little evidence of an acute exacerbation or an acute nephritis. Best's carmine stain demonstrated glycogen in the tubules in the medulla near the cortex and in the loops of Henle.

The AORTA showed moderate sclerosis, no emboli or thrombi were found in any vessels including the coronary arteries which were carefully searched.

Microscopically the aorta showed a thickening of the intimal layers.

HEART: There was a slight excess of fluid in the pericardial cavity.

The heart weighed 385 grams, the ventricles alone weighed 280 grams. The coronary arteries were moderately sclerosed. The aortic valve leaflets showed fresh vegetations, the other valves were negative. The valves measured as follows: Aortic 8 cm. pulmonary 6.5 cm. mitral 10 cm., tricuspid 11 cm.

Microscopically, there was a slight fibrosis throughout the sections. The epicardium was slightly thickened. There were many small collections of polymorphonuclear leucocytes in the myocardium. In a few places there were small colonies of bacteria between the muscle cells. There was some cloudy swelling of the myocardial cells. The endocardium was thickened and heavily infiltrated with polymorphonuclear leucocytes. There was some fibroblastic and angioblastic proliferation, showing that this was a chronic process.

CASE 10—(36-90)

A widower, aged 76, was admitted to the hospital, with the complaint of weakness, enlarged abdomen and itching.

The PRESENT ILLNESS had begun with dyspnoea on exertion, weakness and coughing spells as long as thirty years previous to admission. He had been able to do only light work during this long period but his symptoms had apparently not progressed for about twenty-eight years. Then, that is within two years, his symptoms had developed rapidly. He had suffered an attack of sharp pain in the right upper quadrant which has been followed by swelling of the abdomen, face, legs, ankles and feet. The dropsy had been relieved by drugs. About two months before coming to the hospital oedema of the feet and legs had again appeared. The abdomen had become swollen, the shortness of breath had developed and "smothering attacks" had become troublesome. He had been bedridden for five weeks.

In his PAST HISTORY he had had diphtheria, scarlet fever and tonsillitis. He had been an addict to chronic alcoholism.

The PHYSICAL EXAMINATION showed a sallow, sunken, thin and tightly drawn facies. The skin was pale, dry and excoriated, with oedematous infiltration of the subcutaneous tissue of the back and legs. The costal margins were flaring. There was dullness, diminished tactile fremitus and breath sounds at the bases of the lungs with a few rales near the angles of the scapulae.

The cardiac apex was barely palpable. The point of maximum intensity was felt in the 4th intercostal space. The cardiac dullness extended 10 cm. to the left and 4 cm. to the right of the midsternal line. The second sound in the aortic area was loud and somewhat metallic. The first sound at the apex was replaced by a very loud rasping systolic murmur which was transmitted over the precordium to the axilla. A similar murmur was present in the aortic area and was transmitted to the neck vessels. The blood pressure was 170 mm. mercury systolic and 90 diastolic. The radials, brachials and temporals were beaded and tortuous.

The abdomen was greatly distended with fluid, which rapidly reaccumulated after paracentesis.

The LABORATORY EXAMINATIONS yielded the following results: The urine was negative except for occasional hyaline casts. The phthalein output was 43 per cent in two hours. The blood showed a hemoglobin of 60 per cent, a red blood cell count of 3,240,000 and a white blood cell count of 5,175. The blood Wassermann was negative and the fibrin-lysin test was likewise negative. The blood non-protein nitrogen was 43.9 mg. per 100 c.c. The ascitic fluid was turbid, and straw colored with a low specific gravity and a cell count with a predominance of lymphocytes and nine grams of albumin per liter.

The stools were watery and unformed. Two days before death bright red blood was passed per rectum on three or four occasions. Blood tinged fluid was expectorated. The patient then became comatose, exhibited air hunger and died within 24 hours, that is eleven days after admission. Electrocardiograms showed slight left ventricular preponderance.

The CLINICAL DIAGNOSES were: Alcoholic cirrhosis of the liver, advanced general arteriosclerosis, arteriosclerotic valvulitis, arteriosclerotic nephritis, hypertension, oesophageal varices, starvation acidosis, ectropion, dental caries.

The PATHOLOGICAL DIAGNOSIS were: Thrombosis of the portal and splenic veins, atrophy of the liver with subcapsular fibrosis, chronic mitral and aortic endocarditis, splenomegaly, chronic diffuse, arteriosclerotic nephritis, chronic passive congestion of the intestines, ascites, emphysema, chronic fibrous pleurisy with pericardial adhesions, Meckel's diverticulum, chronic appendicitis, healed pulmonary tuberculosis.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS.

KIDNEYS: The kidneys together weighed 275 grams. The capsules stripped readily leaving slightly irregular surfaces. The cortex appeared thin on section measuring only 2 to 4 mm. in thickness. There was no distinct granulation.

Microscopically there were areas of sclerosis with a moderate number of fibrous glomeruli. There was moderate hyalinization of the walls of the arterioles especially the small ones. The tubules contained a granular precipitate and a few of them contained hyalin casts.

The AORTA was elastic and showed only moderate sclerosis in the arch while the abdominal aorta for 10 cm. above the iliacs showed prominent atheromatous ulceration.

Microscopically the intima was atheromatous and in the muscularis, dark linear bands of calcification were found.

The HEART was contracted. The epicardium over the right ventricle was thickened and white over an area of about 4 cm. A smaller linea area of fibrosis was present over the left ventricle. The mitral valve was incompetent to the hydrostatic test. The mitral leaflets were thickened and deformed. The aortic semilunars were much thickened by granular projections from the upper surfaces. The tricuspid and pulmonary valves were relatively uninvolved.

Microscopically, the endocardium was thick and contained a large number of mononuclear leucocytes with some small foci of polymorphonuclear leucocytes. The subendocardial connective tissue was slightly increased in amount. The myocardium showed a very marked mononuclear infiltration with some foci of polymorphonuclear leucocytes surrounding small colonies of blue staining granular masses of bacteria. There were also some areas of calcification in the myocardium. There was a marked angioblastic and fibroblastic proliferation throughout the sections. In the muscle near the epicardium there were also some small colonies of cocci. There was a chronic passive congestion throughout all the sections. Some of the muscle fibers showed simple necrosis but the majority showed only a marked degree of cloudy swelling.

CASE 11—(7-7)

An unmarried negro, age 48, was admitted with the complaint of shortness of breath, pain over the heart, and a sensation of fullness of the stomach.

The FAMILY HISTORY was irrelevant except that the patient's father had died of "Bright's Disease."

In his PAST HISTORY he had had gonorrhea several times, but denied ever having had syphilis.

The PRESENT ILLNESS had begun rather suddenly about two months before admission when he was awakened one morning by a shortness of breath, which he never had had previously. He also had a sensation of fullness in the epigastrium, but no pain. Within three days he had to stop working because of weakness and dyspnoea. The symptoms had increased progressively. The week previous to admission he had suffered from severe pain in the upper chest and over the base of the heart, when he reclined especially on his left side. This had disturbed his sleep, but had not inconvenienced him much when he was up and about erect during the day. The dyspnoea, however, had been more troublesome during the day. The feet and ankles had been swollen at night during the first week of symptoms. He had had a slight cough, but he had produced very little sputum; and no blood. His appetite had remained good and all foods could be taken with no symptoms except for the occasional production of distention. He had been chronically constipated. He had had nocturia 7 to 8 times each night, with the production of large quantities of urine at each voiding. He had had some dribbling 4 and 5 times a day.

The PHYSICAL EXAMINATION showed a drowsy dyspnoeic negro with puffiness of the face and dry skin. The pupils were contracted and fixed probably from morphine effect, but reacted normally at later examinations. The teeth were dirty and some pyorrhea was present. The tonsils were enlarged.

The neck veins were conspicuously engorged and there was visible and palpable throbbing over the carotids and subclavians. The cervical glands were palpable and shotty.

On percussion note over the chest was resonant except for impairment at the left base. In this same region the tactile fremitus was absent, the breath sounds were diminished and egophony and rales were heard.

The heart pulsation was widespread over the precordium, but fairly forcible. The apex impulse was located with slight difficulty in the 6th intercostal space 13 cm. to the left of the midsternal line. An intense diastolic thrill was felt over the upper sternum extending 4 cm. to the left of the midsternal line as far down as the third rib; on the right side it was felt from the first rib down to the fifth rib. The retromammary dullness measured 9 cm., while the cardiac dullness extended 4.5 cm. to the right and 13.5 cm. to the left of the midsternal line.

Corresponding to the long diastolic thrill a long musical diastolic murmur was heard over the entire anterior aspect of the chest and well into the axillae, replacing the aortic second sound. The aortic first sound was not heard. A blowing systolic replaced the mitral first sound at the apex, but the murmur was not heard transmitted toward the axilla. The mitral second sound was replaced by the transmitted aortic diastolic murmur. The blood pressure was 180/75 and 150/50. The pulse was regular and of a poor Corrigan type. There

was a faint pistol shot sound but no definite diastolic murmur in the compressed femoral artery.

The abdomen was distended and the liver was tender and extended 6 cm. below the costal margin. Definite pulsation was felt over the liver region, but it was not expansile. The spleen was not felt.

The reflexes were normal. The peripheral arteries were thickened, but not calcareous. The feet and legs were oedematous.

The LABORATORY EXAMINATIONS showed: The urine volumes were low with specific gravities of 1018 to 1032. The reaction was acid. Albumin in amounts of 0.5 to 1.0 gram per liter was present. Hyalin and granular casts, but no leucocytes or erythrocytes were present in the sediment. The 'phtalein excretion was 50 per cent in two hours, but later dropped to 35 per cent.

The blood examination showed 70 per cent hemoglobin, 4,500,000 red blood cells, and 7,500 to 10,500 white blood cells. The differential count was not abnormal to any recognizable degree. The Wassermann reaction was repeatedly negative. Blood cultures were negative. The blood N. P. N. was 43 mg. and later 51 mg. per 100 c. c. The pleural fluid was serosanguinous with a specific gravity of 1012, an albumin content of 1 gram per liter, and 2100 lymphocytes per c. mm. The chest X-ray showed cardiac enlargement, passive congestion of the lungs and a left hydrothorax. Electrocardiograms showed moderate left ventricular preponderance.

COURSE IN THE HOSPITAL: The aortic diastolic murmur lost its musical character and a systolic aortic murmur appeared. The retromammary pain persisted and was quite severe. The oedema and congestion were not relieved by the Karell treatment and digitalization to the point of producing auricular fibrillation. The transient irregularity disappeared in a few days after the drug was discontinued. The apex beat became palpable in the 6th intercostal space 13.5 cm. to the left of the midsternal line. An irregular fever was constantly present.

The electrocardiograms recorded left ventricular preponderance, ventricular extrasystoles, a transient auricular fibrillation and the reestablished normal mechanism with occasional interpolated extrasystoles.

The patient was under observation until his death two and one-half months after his admission.

THE CLINICAL DIAGNOSES were: Chronic cardiac valvular disease, aortic insufficiency. Acute endocarditis. Cardiac failure with congestion. Chronic myocarditis. Cardiac hypertrophy and dilatation. Chronic nephritis. Chronic general adenitis. ?Syphilis. Arteriosclerosis.

THE PATHOLOGICAL DIAGNOSES were: Acute and chronic vegetative aortic, mitral and tricuspid endocarditis. Chronic aortitis. Pulmonary infarct with oedema. Chronic orchitis. Arteriosclerotic nephritis. Chronic passive congestion of the viscera. Epicarditis (soldier spot). Chronic fibrous pleurisy and calcified tuberculous peribronchial lymphadenitis.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS:

KIDNEYS: Each weighed 120 grams and measured 12x6.5x3 cm. The capsules stripped with slight difficulty and the exposed surfaces were red and finely granular. On section the normal kidney markings were found distorted in places by scars. There were sclerotic vessels in the subcortical zone, with thickened walls and patent lumina projecting conspicuously above the level of the cut surface.

Microscopically, there was an advanced arteriosclerosis associated with the usual scars. Hyalin casts were present and an occasional dilated tubule was filled with eosinophilic homogenous material.

AORTA: The base showed a few small pin point plaques of atheromatous degeneration. The intima appeared wavy when covered with a thin film of blood. Microscopically, there were atheromatous changes in the intima and media.

HEART: The organ was much enlarged. The coronary veins were greatly congested. A few epicardial soldier spots were seen. Vegetations were numerous on the aortic, mitral and tricuspid valves. On the aortic and mitral valves, the process was mainly on the ventricular side, while on the tricuspid there were especially prominent lesions on the auricular surface. All the valve margins were thickened. Microscopically, the valves showed thickenings and mononuclear infiltration. At one place a mass of granulation tissue with abundant polymorphonuclear infiltration adhered to the valve. In other places fibrin vegetations adhered to the valve surfaces.

Microscopically, all the sections of the myocardium showed increase in connective tissue. In some areas there was a slight mononuclear leucocytic infiltration which was not accompanied by any evidences of chronicity, such as angioblastic and fibroblastic proliferation. The epicardium was little affected. The endocardium was slightly thickened and in places showed a leucocytic infiltration. Certain areas of the sections showed advanced cloudy swelling of the muscle cells.

CASE 12—(9-9)

A man, aged 45 years, was sent into the surgical ward as an "acute abdomen" because of sharp pain in the epigastrium slightly to the right of the mid-line.

In his FAMILY HISTORY his father had died at 69 of uræmia, his mother at 47 of marasmus, and two brothers of pneumonia. He had been married since 1900, but had been separated from his wife for the last eight years. There had been no children and no miscarriages.

In his PAST HISTORY he had contracted gonorrhea at 21. A soft chancre also appeared 7 days after the exposure and disappeared in 3 weeks. He had had a questionable chancre at 24 with a secondary rash. At 30, he had had an attack of sharp pain in the back, which did not radiate, but "doubled him up" and was followed by "jaundice." A physician diagnosed the trouble as gall stone colic and relieved the pain by hot applications.

He had indulged in 4 to 5 glasses of beer and a half glass of whiskey, 3 to 4 cups of coffee, and a third of a package of smoking tobacco per day. He had lost 20 pounds of weight within a year.

He had been struck over the vertex one month previous to admission. Erysipelas had developed and an alopecia had resulted. He had noted a soreness in bones and joints and felt generally below par after that.

His PRESENT ILLNESS, in his opinion, however, had begun suddenly when he was awakened from a sound sleep at 2 A. M. the day of admission, with a pain in the epigastrium a little to the right of the mid-line. It "cramped him into a knot", did not radiate, but remained localized, steady, and sharp. The pain was less intense when he lay on his back, but rolling around was necessary to ease it entirely. About one hour after the onset, he began to vomit, bringing up at first the food taken at supper, then a bad-smelling brown material, but no blood so far as he knew. He vomited everything taken by mouth after that. There remained a dull pain or soreness in the epigastrium. He had no definite chill or fever. His bowels were moving regularly. The pain gradually increased in intensity until it was very severe. The pain came in paroxysms, especially after pappation or movement.

The PHYSICAL EXAMINATION showed a large fairly well-built man, with muscles of good size. He was distinctly pale and sallow. The panniculus was moderately thick. There was an alopecia over the occipital region. The pupils reacted to light and accommodation. There were masses of follicles on the left lateral aspect of the tongue, one centimeter anterior to the glossopalatine junction. The follicles were inflamed, enlarged, covered by a diphtheroid membrane, simulating a late secondary luetic lesions. The tip of the tongue presented a fissure of the same general appearance. A few PETECHIAL MARKINGS WERE NOTED on the pharynx. The teeth were poor, a few were missing, and there was a moderate pyorrhea. The neck was negative except for palpable cervical glands.

The lungs were resonant and clear throughout. The heart apex was in the 5th intercostal space, 10 cm. to the left of the midsternal line and 4 (?) cm. to the right. The heart rate was 115 and the rhythm was regular. An embryocardia was noted and systolic murmurs were heard in the aortic and mitral areas. The pulse was small and regular. The blood pressure was 165/90 and 160/90.

The abdomen was scaphoid. There were no visible masses or peristalsis. Slight tenderness was noted in the right upper quadrant. There was some resistance in the epigastrium during the paroxysms of pain. The spleen was not palpable, but there was some resistance and tenderness in the region. The liver dullness extended 1 cm. below the costal margin, where the edge was also felt. No fluid wave or shifting dullness were elicited.

The extremities showed no edema, ankylosis, or tenderness. The T. P. R. were respectively 99.8, 130, 24.

The LABORATORY EXAMINATIONS showed blood in the sputum, vomitus and stools. The stools were very foul and contained much blood and many trichomonads. The hemoglobin was 70 per cent and the red cells numbered 3,940,000, while the leucocytes were 14,000. The urine showed a slight trace of albumin, a few red and white blood cells, but no casts. The blood Wassermann was a 2-plus with the cholesterinized antigen. The electrocardiogram showed no preponderance.

COURSE IN THE HOSPITAL: Occasional shooting pains were noted in the joints and in the subdeltoid bursa, and the inner condyle of the right humerus became painful. A group of petechiae (75-100) appeared between the 8th and 10th ribs. The lesions varied in size and were copper-red in color along the 9th intercostal space. The clotting time, bleeding time, and platelet count were normal.

The patient was transferred to the medical ward, as the petechial spots became generalized. Blood cultures remained sterile. The petechiae became more like purpura. Subcutaneous hemorrhagic areas then appeared over all the joints and pressure points. The areas faded and recurred. The clotting time was $3\frac{1}{4}$ minutes. The bleeding time was prolonged to 10 minutes. The platelets were reduced in numbers to 52,000.

The cardiac findings did not change, but an increase in the hemorrhagic areas and the marked pallor and a tinge of cyanosis were noted. There was a deeply sloughing ulcer on the tongue and palate. The gums were hemorrhagic and inflamed and exuded pus on pressure. There was a consolidation of the whole right lung and lower lobe of the left, which was considered a pneumonia rather than the results of infarction. The temperature rose to 101 degrees and the leucocytes numbered 22,000 to 25,000.

The patient became more and more toxic and died seven days after admission.

The CLINICAL DIAGNOSES were: purpura hemorrhagica, lobar pneumonia of the right lower lobe, edema of the lungs, and (?) syphilis.

The PATHOLOGICAL DIAGNOSES were: purpura of the skin, mucous membranes of the mouth, conjunctiva of the right eye, and the large and the small intestine. There was ulceration of the duodenum, the upper jejunum, and the lower part of the ileum. Localized fibrino-purulent peritonitis. Enlargement of the spleen with multiple hemorrhages. Acute lobar pneumonia on the right. Thrombosis of the right pulmonary artery. Acute local pulmonary edema on the left. Lung cultures contained Group IV pneumococci and B. influenzae.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS:

KIDNEYS. The left kidney weighed 190 grams and measured 11.5 by 6.5 by 4.3 cm. The right weighed 205 grams and measured 12.5 by 7.5 by 4 cm. The capsules were thin and adherent in several places and tore when being stripped. The cortex was otherwise smooth and had a grayish-red color and measured 7 mm. in thickness. In some places the striations could be easily made out; in others they were rather indistinct. The medullae were slightly congested.

Microscopically the parenchyma cells were swollen and granular and in places showed slight necrosis. The glomerulae were distended. The cells lining Bowman's capsule were small and granular. The convoluted tubules were slightly distended and contained pink-staining granular material in small amounts.

AORTA: The base was thickened with tough fibrous areas which involved the entire thickness of the arterial wall. The coronary orifices stood widely open. Microscopically there were atheromatous areas in the media and intima.

HEART: The organ weighed 305 grams. Fibrous bands extended from the anterior wall of the right auricle to the lower margin of the coronary sinus. The foramen ovale was patent, but a flap covered part of the opening. The pulmonic leaflets were fenestrated, the other valve leaflets were thin. No vegetations were noted. The valves measured: Aortic, 8 cm., pulmonary, 8 cm., mitral 11 cm., tricuspid 13 cm.

A subendocardial hemorrhagic area was noted at the top and left side of the interventricular septum, in the region of the branches of the His bundle.

Microscopically the muscle fibres were hypertrophied. The epicardium showed no conspicuous changes except in one place along the atrioventricular groove in the left ventricular wall where there was a heavy infiltration of polymorphonuclear leucocytes in the myocardium, subepicardial fat and connective tissue. There was an increase in connective tissue about this chronic suppurative focus. The endothelium showed some thickening in various areas.

CASE 13—(64-149)

A woman, aged 48, was admitted to the hospital with the complaints of pains in the legs, incontinence, weakness and a cough.

Her family history and past history were irrelevant.

Her **PRESENT ILLNESS** had begun insidiously about six months before admission with weakness. She had contracted a severe cold that had been diagnosed "influenza" in March 1920. During the first week of the illness there had been persistent vomiting. She had remained in bed for a month, on getting up she found that her feet and ankles had swollen. This oedema remained for two months. About five weeks before admission she had taken to bed because of severe lightning pains in the legs, which had come on suddenly, two weeks previously. Incontinence had been noted for four weeks. For two weeks before admission morphine had to be used for the pains. The patient had always been delicate and never stout, but still had lost much weight during her illness.

The **PHYSICAL EXAMINATION** showed emaciation, and cachexia, with a yellowish tint to skin. Four large deep decubitus ulcers were present over the sacrum and lower back. The lungs here and there throughout showed broncho-vesicular breathing and rales. The abdomen was negative. The reflexes were all hyperactive. There was ataxia in the arms and legs and a bilateral positive Babinski reaction. There were marked involuntary flexor spasms and paraesthesias noted.

The **LABORATORY EXAMINATION** showed: Hemoglobin 20 per cent with R. B. C. 1,172,000 and W. B. C. 4,400. Many oval forms of red cells, anisocytosis, slight polychromatophilia and few platelets. The Wassermann was negative. The urine contained pus and cultures showed *B. Coli*. Electrocardiograms showed no preponderance.

COURSE IN HOSPITAL: There was no improvement. Fever was present each afternoon. The pulse was rapid, from 100 to 162. Attacks of dyspnoea appeared. The patient was irrational at times. The pulse gradually grew weaker. The thready pulse became imperceptible. Rhonchi were heard in the lungs. Unconsciousness intervened. The patient died eleven days after admission.

The **CLINICAL DIAGNOSES** were: Pernicious anemia, posterior and lateral spinal sclerosis, bronchopneumonia, chronic cystitis and decubitus ulcers.

The **PATHOLOGICAL DIAGNOSES** were: Pernicious anemia, posterior sclerosis of the cord. Bronchopneumonia of the left lower lobe. Pulmonary oedema and emphysema. Hydropericardium, left hydrothorax, ascites. Chronic diffuse nephritis. Atheroma of the aorta. Anomaly of the coronary artery. Caseous and calcified tuberculosis of the mediastinal and peribronchial lymph nodes. Bilateral pleural and pleuropericardial adhesions. Nodular thickenings of the epicardium, liver capsule and peritoneum of broad ligament. Blood cultures showed hemolytic streptococci and staphylococci.

GROSS AND MICROSCOPICAL DESCRIPTIONS.

KIDNEYS: The kidneys together weighed 140 grams. The right measured 9.5x5x3 cm., the left 11x5.5x3. The capsule stripped with difficulty leaving torn surfaces and tearing itself. The surfaces were deep reddish brown in color and distinctly granular. On section the parenchyma was opaque and pale. The pelvis showed marked infiltration. The cortex was 4 mm. in diameter in places.

Microscopically there were numerous hyalin casts in the collecting tubules, many of which also contained a granular sediment. There was an increase in interstitial tissue. In places there was a round cell infiltration which involved the glomeruli, some of which had undergone degeneration. The capsule was thickened.

AORTA. Fibrous yellowish white patches were present throughout the vessel with yellowish nodular thickenings at the orifices of the branches. The branches showed no apparent change. The aorta was elastic.

Microscopically the aortic intima was thickened by layers of fibrous tissue.

HEART. There was but one coronary artery origin, the orifice of which was 1 cm. above the aortic cusps. The common coronary gave origin to the left and right branches almost immediately. The pericardium contained 100 c. c. of clear straw colored fluid. There were a few pleuropericardial adhesions.

The **HEART** weighed 285 grams, was gray brown, soft and flabby and contained very little blood. The epicardium showed two large soldier spots. The muscle bundles showed distinctly the banded subendocardial fatty degeneration. The valves measured as follows: aortic 7 cm., mitral 10 cm., pulmonary 7 cm., and tricuspid 14 cm.

Microscopically the cardiac muscle fibers were large and had large oval nuclei, much larger than usual. Many cells showed an advanced fatty degeneration. There was an extremely large number of fibroblasts and some leucocytes throughout the sections. The blood vessels showed some acute passive congestion, the walls were but moderately thickened. The epicardium and epicardial fat were slightly thicker than normal in places. The endocardium was thickened in certain areas.

CASE 14—(6-6)

A male, aged 57, was admitted with symptoms of prostatism. Convulsive seizures came on shortly after admission.

His family, marital and past histories contained no significant facts.

The PRESENT ILLNESS had begun with a pain in the suprapubic region and a general "tired feeling". Frequency of urination and dysuria and hematuria had been noted within a year. The symptoms had been worse in attacks at intervals of a few months. The frequency and nocturia had gradually become much worse and an intense thirst had been noted. At the same time drowsiness, mental confusion and puffiness of the face had developed. There had been a loss of 30 pounds of weight.

Shortly after admission the patient had three generalized convulsions which were followed by transient signs of right hemiplegia, bilateral Babinski reactions, and exaggerated reflexes.

The PHYSICAL EXAMINATION in general revealed sclerosis of the peripheral arteries.

The pupils were small, slightly irregular and reacted sluggishly to light. The disc borders were slightly hazy.

The lungs were hyperresonant throughout, except at the right base where there were signs of fluid. Rales of all types were heard throughout the chest. The breath sounds were puerile in character.

The heart was apparently slightly enlarged 4 cm. to the right and 12 cm. to the left of the midsternal line. The aortic second sound was accentuated and a slight systolic murmur was heard at the apex. The blood pressure was 210/110 during the convulsions.

The abdomen contained a large smooth immovable slightly tender mass under the right costal margin. Pressure upon this mass caused a flow of urine after the bladder had been emptied by a catheter. Rectal palpation revealed an enlarged hard nodular prostate. The reflexes were hyperactive. Bilateral Babinski reaction and ankleclonus were present. The signs of the hemiplegia rapidly disappeared.

The LABORATORY EXAMINATIONS showed: 70 per cent hemoglobin, 3,584,000 red blood cells and 28,400 leucocytes. The blood non-protein nitrogen was 215 mg. per 100 c. c. but fell to 166 mg. on the following day. The Wassermann reactions on the blood and spinal fluid were negative.

The electrocardiogram showed a questionable left ventricular preponderance, more pronounced in the curve taken immediately following a convulsion.

COURSE IN THE HOSPITAL: After the convulsions, which subsided following blood letting and lumbar puncture, the patient lingered along with bladder drainage accomplished through a permanent catheter. The neurological signs disappeared. The blood pressure dropped to 180/120 then 160/98 and finally to 110/70. The blood non-protein nitrogen gradually rose to 246 mg. per 100 c. c. The asthenia and drowsiness progressed to stupor and coma in which the patient died three weeks after admission.

The CLINICAL DIAGNOSES were: Carcinoma of the prostate, chronic uremia, chronic nephritis, arteriosclerosis, hydronephrosis, chronic cystitis, emphysema, retention cyst of the scrotum.

The PATHOLOGICAL DIAGNOSES were: Adenocarcinoma of the prostate with extension into the posterior and upper wall of the urinary bladder and lymph glands along the left ureter and base of the mesentery and a metastasis to the right pleura. Bilateral hydronephrosis and hydroureter. Chronic interstitial nephritis and myocarditis with calcium deposits. General arteriosclerosis. Chronic fibrous pleurisy and oedema of the lungs. Sebaceous cyst of the scrotum.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS.

KIDNEYS: The left kidney weighed 260 grams, while the right weighed only 130 grams. The capsules stripped with some difficulty, exposing roughened scarred surfaces. The pelves and ureters were dilated, the dilatation extended well up between the pyramids. The functional tissue of the kidneys was greatly decreased in amount and there was an evident increase in connective tissue. Microscopically, there was a diffuse infiltration of connective tissue, with degeneration of tubular epithelium.

AORTA. The elasticity was greatly decreased. The intimal surface showed many raised yellow patches of atherosclerosis. Microscopically, there was connective tissue infiltration with some hyalinization and calcification. The coronary arteries were extensively calcified.

HEART. The heart weighed 315 grams and appeared normal externally. The muscle was dark red and showed only one abnormal area which was situated in the wall of the left ventricle posteriorly about 2 cm. from the apex. There was a darker harder part of the muscular wall. The valve leaflets were slightly thickened, but not otherwise abnormal.

Microscopically, sections of the myocardium showed a decided increase in connective tissue with atrophy of some of the muscle fibers. In many areas of increased connective tissue there were extensive deposits of calcium. The muscle fibers were hypertrophied, and between them there was a rather large amount of connective tissue. The subepicardial fat was large in amount and stained more bluish than normal.

CASE 15—(10-10)

A bachelor, aged 59, was admitted with the complaint of cramps in the right wrist and arm and also in the right half of the face and in the right leg.

The family history was irrelevant.

The PAST HISTORY was significant in that he had had recurrent attacks of glycosuria since the age of 17.

The symptoms that he had complained of at his first admission to the hospital had been cramps in the face and the right wrist, extending to the fingers and to the shoulder, also in the right leg. His first attack had occurred two years previously and had consisted in the hand getting cold before the attack and remaining so for ten minutes. There had been marked blanching in the hand before the attack, then after the coldness there came a gradual return of warmth and color. The attacks came as often as 4 or 5 a day and

were accompanied by dizziness. He had lost 20 pounds in weight in the month before admission.

The PHYSICAL EXAMINATION showed emaciation, amblyopia in the right eye, due to traumatic corneal scar and adhesive iritis. The left pupil was small and fixed. The arteries were thick and tortuous. The chest was hyperresonant. The heart borders were not made out and no murmurs were heard. The blood pressure was 115/90. The arm reflexes were absent. The right knee jerk was greater than the left. No pathological toe signs were found. The Romberg was positive. Cystoscopy showed a neurogenic bladder.

The LABORATORY STUDIES showed: Blood sugar .130 per cent; blood non-protein nitrogen 69 mg. per 100 c.c. The 'phthalein excretion was 20 per cent in two hours. He excreted a trace to 12 grams of sugar per day. Occasionally he was sugar free on higher diets. The NH₃N was low. He gained 2 pounds—from 118 to 120. He was sugar-free on discharge and had been so most of the time.

Six months later, he was admitted to surgery for a furunculosis and transferred to metabolism for treatment. The blood sugar was 0.3339 per cent and the blood N. P. N. was 28 mg. per 100 cc. The 'phthalein output was 60 per cent in two hours. His blood pressure was 135/65. His weight was 122 pounds. A large open sloughing boil in the left buttock, with an area of induration 11 cm. in diameter, was found. This was a carbuncle. Treated locally the lesion healed rapidly.

Three months later he was admitted to Surgery for a Bromic acid burn of the lateral part of the thigh and right leg. He was transferred to metabolism with an impending coma.

The PHYSICAL EXAMINATION showed: An extensive second degree burn of the outer side of the right leg, extending from the mid-thigh to the ankle. The panniculus was thin, the muscles were wasted, the skin was dry, rough, and loose.

HEART: No shock or thrill were noted. The cardiac outline was within normal limits. Numerous extrasystoles disturbed the rhythm. The aortic second sound was accentuated. No murmurs were heard.

LUNGS: The respiratory excursion was fair; there was no lagging. The percussion note was resonant throughout. The breath sounds were bronchovesicular. The whispered and spoken voice were normal. Occasional rhonchi were heard.

EXTREMITIES: The brachials and radials were thickened and tortuous. The blood pressure was 140/70.

The LABORATORY EXAMINATIONS showed: Sugar and diacetic acid in the urine, but no albumin or casts. The blood CO₂ was 54.1; blood sugar 0.36 per cent and non-protein nitrogen 36.2 mg. per 100 cc.

The blood examination showed 70 per cent hemoglobin, 4,080,000 red blood cells, and 14,800 to 30,400 leucocytes. The differential count of a smear was: polymorphonuclears 84 per cent, large mononuclears 10 per cent, and lymphocytes 6 per cent. Electrocardiograms showed extrasystoles but no preponderance.

COURSE IN THE HOSPITAL: The T. P. R. chart showed an irregular fever, T. 104-100 degrees; P. 120-100; R. 20-44. Hiccough developed and became troublesome. Sugar and diacetic acid appeared in the urine in increasing amounts. The patient gradually lost ground. An amputation was done as a last resort to stay the downward progress, but the shock was too great and a terminal coma developed.

The CLINICAL DIAGNOSES were: Diabetes mellitus. Burn of right leg. Post operative wound infection. Abscess of right lung. Furunculosis. Arteriosclerosis. Syphilis of the cerebrospinal meninges. Neurogenic bladder. Amblyopia.

The PATHOLOGICAL DIAGNOSES were: Amputation infection. Abscess of upper lobe of right lung. Arteriosclerosis. Chronic nephritis. Chronic interstitial pancreatitis. Calcification of spinal dura mater. Partial obliteration of cerebral convolutions. Opacity of the right cornea with atrophy of the eye.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS.

KIDNEYS: The right kidney weighed 150 grams and the left 155 grams. They were small and hard. The capsule stripped off with some difficulty, leaving exposed a rough pebbled surface, typical of the small red granular kidney.

Microscopically there were scars densely infiltrated with mononuclear cells scattered throughout. Tubules caught in the scars were either entirely obliterated or showed various phases of destruction, being filled with hyaline casts and granular detritus.

AORTA: The elasticity was entirely lost. Wide-spread atheromatous degeneration was noted. Microscopically the sclerosis involved the media and the intima. The coronaries were thickened and tortuous.

HEART: The heart weighed 300 grams and presented no abnormalities on the gross inspection of the epicardial and endocardial surfaces. The valve leaflets were slightly thickened, but no lesions sufficient to produce disturbances to the flow were present.

Microscopically there was a conspicuous increase in connective tissue, a marked fibrosis. The muscle cells were smaller than normal. The subendocardial fat was abundant. The endocardium showed no changes.

CASE 16—(31-95)

A widow, aged 75, was admitted, in a semi-comatose condition with the complaints of pain in the feet and gangrene. Delirium and disorientation made it difficult to obtain a history. Her son gave the following history:

The FAMILY HISTORY was of interest in that one sister and one brother had had cancer. The PAST and MARITAL HISTORIES were negative.

The PRESENT ILLNESS had begun about six months before admission with pains in the feet which persisted and were aggravated by walking, and later by mere touching, and finally there was a pain at rest also. About two weeks before admission the little toe on the left had become dark, and with this the pain had become excruciating. Morphine was re-

quired. Queer actions and a generally semistuporous condition had been present for ten days previous to admission.

The PHYSICAL EXAMINATION showed an evident comatose condition, dehydration and emaciation. There were carious teeth, and infected gums and enlarged glands in the neck. The lungs were hyper-resonant. The breath sounds were increased over the back and a few rales were present at the end of inspiration. The heart was not enlarged. A soft blowing systolic murmur was heard at the apex transmitted into the axilla. The B. P. was 200/70 on admission, 160 to 180/70 for a week, then followed a gradual drop to 130/40. The blood vessels were markedly sclerotic and tortuous. The extremities showed dry black atrophic skin over all the toes with lines of demarcation over the dorsa of the feet. No pulsations were felt in the dorsalis pedis arteries. The fingers were cyanosed. A vaginal examination was reported negative.

LABORATORY DATA: The urine contained albumin, casts, r. b. c. and w. b. c. The 'phtalein excretion was 44 per cent. The blood showed a slight secondary anemia. The CO₂ Vol pr cent was 71. The blood sugar per cent was 11. The blood non-protein nitrogen was 40 mg. per 100 c.c. Electrocardiogram showed no perponderance.

COURSE IN THE HOSPITAL: The patient was given hypodermoclysis and rectal taps of glucose. She moaned whenever slightly disturbed, and was irrational and disoriented most of the time. She was given 900 c. c. of saline intraperitoneally. She developed a swelling in front of the left ear. Rales and harsh breathing appeared in the chest. The leucocytes rose from 10,000 to 52,000. Glucose intervenously was of no avail. The pulse gradually became weaker, and the patient died three weeks after admission.

The CLINICAL DIAGNOSES were: Endarteritis obliterans with gangrene of the feet. Arteriosclerosis general. Abscess of the parotid. Hallux valgus.

The PATHOLOGICAL DIAGNOSES were: Gangrene of the feet. Arteriosclerosis. Acute parotitis. Carcinoma of the cervix uteri with metastases to the hypogastric and retroperitoneal lymphnodes. Volvulus of the caecum and ascending colon. Septicaemia with petechiae of the skin. Chronic aortic and mitral endocarditis. Chronic pancreatitis. Duodenal diverticulae. Healed tuberculosis of the left lung, peribronchial glands and spleens.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS.

KIDNEYS: The right kidney weighed 100 gm., the left 95 gm. The capsules tore on attempted removal bringing away some of the cortex. The surface was somewhat granular. On section the kidney was pale and showed a few opaque areas. The cortex appeared narrow. Microscopically there was a conspicuous increase in interstitial fibrous tissue. In places there were small accumulations of lymphocytes. The cytoplasm of the cells of the tubules was granular and there was debris within the tubules. The glomeruli contained an increase of polynuclear cells. The arterioles showed moderate sclerosis.

AORTA. The aorta showed a very extensive sclerosis with hard calcified plaques throughout its course. Microscopically there was a marked thickening of the intima with calcareous plaques and cholesterol crystals. There was a slight cellular infiltration about several of the vasovasi in the media. The coronary arteries showed considerable sclerosis. Microscopically there was intimal thickening with some calcification and narrowing of the lumina.

HEART. The heart weighed 280 grams and appeared fairly normal. Hydrostatic tests showed a slight tricuspid insufficiency. The valves measured as follows: aortic 8 cm., pulmonary 8 cm., mitral 10 cm. and tricuspid 13 cm. The aortic valve was thickened and contained a hard calcified nodule in one cusp, similar to those in the aorta just above the valve. The mitral leaflets were thickened and sclerosed.

Microscopically there was a moderate increase in fibrous connective tissue throughout the sections. The muscle fibers were for the most part smaller than normal. The blood vessels were markedly thickened and congested. There was a slight acute leucocytic infiltration of the myocardium. The epicardium and endocardium showed no changes.

CASE 17—(48-86)

A colored female, aged 37, was admitted with the complaint of pain in the left foot and leg.

No FAMILY HISTORY of significance was obtained. She had one spontaneous abortion at the 3rd month.

Her PAST HISTORY was indefinite. Four months previously she had had a sense of fullness in the stomach, with nausea and occasional vomiting.

Her PRESENT ILLNESS had begun insidiously about a month before admission with fever, weakness, general malaise, anorexia, nausea and a sensation of heaviness in the stomach with no belching and only occasional vomiting. The patient had remained up and about. A week before admission there had been a sudden onset of high fever, profuse sweating and prostration. During this attack she had panted for breath and had had to be propped up in bed. She had had a severe chill following which all parts of the body recovered warmth except the left leg and foot which remained cold and painful.

The PHYSICAL EXAMINATION showed apathy, tachypnoea and slight disorientation. The tongue was coated; teeth were carious; gums were retracted and contained infected pockets. The tonsil crypts were filled with pus. The cervical glands were palpable. The Thorax: Respirations were shallow at a rate of 35 per minute. A to and fro leathery sound, close to the ear, was heard in the right axilla. The percussion note was dull from the angle of the right scapula to the base, and also impaired to a less degree at the left base. The breath sounds were diminished at the right base. Bronchial breathing and crepitant rales were heard at the left base. The heart was 3 cm. to the right, and 10 cm. to the left. The aortic dullness was 6 cm. The heart sounds were regular rapid and distant. The pulmonary second sound was slightly accentuated. The blood pressure was 116/65.

The abdomen was rigid all over. No tympanites was present. No obliteration of the liver dullness was noted. General tenderness was present throughout. The pelvis was negative. The left foot and leg were black, wrinkled and dry except for a few large blebs anteriorly. The limb was insensible to pin prick up to a sharp irregular line of demarcation from normal skin 10 cm. below the knee. There was hyperalgesia above the line. No

pulsation was felt in the leg, and no warmth was present. There was slight oedema above the knee on the left.

THE LABORATORY DATA SHOWED: The urine contained albumin in large amount, numerous coarse granular casts, w. b. c. and a few r. b. c. Urine culture showed *B. Coli*. The blood showed 60 per cent hemoglobin, 3,650,000 erythrocytes, 13,200 to 25,000 leucocytes. The Wassermann was negative. Blood cultures were negative. The sputum contained blood and some streptococci. Stool cultures showed Paratyphoid *B*. The Widal was negative. The chest fluid showed 23,800 cells 40 per cent r. b. c. 40 per cent polymorphonuclears; 4 per cent epithelial cells. 3.5 per cent nor normoblasts ? and 1 per cent large mononuclears. No mitotic figures and no organisms were present. Electrocardiograms showed moderate left ventricular preponderance.

The patient gradually lost ground and died 17 days after admission. The gangrenous leg had become more and more putrid.

THE CLINICAL DIAGNOSES were: Paratyphoid *B* fever. Pulmonary infarcts. Chronic pulmonary tuberculosis? Right pleural effusion. Chronic nephritis. Thrombosis of the left popliteal artery with gangrene of the leg.

THE PATHOLOGICAL DIAGNOSES were: Thrombosis of the uterovaginal and ovarian plexuses bilaterally, of the left ovarian vein and embolism of the left external iliac and popliteal arteries. Dry gangrene of the left leg. Cardiac hypertrophy and dilations. Mural thrombi in the left ventricle. Acute vegetative mitral and tricuspid endocarditis. Multiple infarcts of the lungs and spleen. Right fibrinous pleurisy. Chronic pancreatitis. Chronic nephritis. Cultures yielded *B. coagulans*.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS

KIDNEYS: The right kidney weighed 140 grams and measured 12x6x2.5 cm., the left 105 grams and measured 12x7x3 cm. The capsule stripped readily. Cloudy swelling and some small gray white opaque areas were noted in both kidneys. Microscopically there was an acute tubular nephritis. The tubular epithelium in many places was swollen and granular cells were desquamated and collected in the lumina. There was a granular sediment within the tubules and also within Bowman's capsule. Some of the tubules showed various infoldings as if the epithelium were hyperplastic. The cells were hyperchromatic in these areas. The interstitial tissue was oedematous and there was considerable congestion. There were numerous hyalin casts. Some tubules contained collections of polymorphonuclear leucocytes. The small arterioles showed sclerosis. Fat stains showed advanced fatty degeneration of the tubules.

HEART: The heart was distinctly enlarged, weighing 480 grams. The hydrostatic test showed a tricuspid insufficiency which measured 2x3 cm. The mitral showed a slight insufficiency which measured 2.5 mm. across. The valve circumferences were as follows: tricuspid 12 cm., mitral 10.5 cm., pulmonary 6 cm., and aortic 7 cm. The pulmonary valve flaps showed some fine fenestrations while the aortic leaflets were negative. The tricuspid and mitral segments showed some small red nodules which were probable vegetations. The foramen ovale had a small 1 mm. slit-like opening in it.

On the anterior surface of the left ventricle just to the right of the papillary muscle was a smooth flat mural thrombus irregular in shape measuring about 4 x 2 cm. The adherent mass was white and contained a smooth lined cavity. Another irregular mass 1.5x2 cm. was found behind the anterior papillary muscle.

Microscopically the valves showed no changes. Throughout the sections were areas of dense leucocytic infiltration. There was a marked fibroblastic and angioblastic proliferation. Areas in which the muscle fibers were entirely gone showed partial replacement by connective tissue. In some sections a mixed clot was found firmly attached to the endocardium, a parietal or mural thrombus. The blood vessels were thicker than normal especially in sections taken from near the atrioventricular groove. The myocardium showed advanced cloudy swelling in areas in which the leucocytic infiltration was not so marked.

CASE 18—(41-85)

A man, aged 50, was admitted in a state of coma. His previous complaints had been pain in abdomen, nausea and vomiting.

The **FAMILY, MARITAL** and **PAST** histories contained no significant facts. Information was obtained from friends.

The **PRESENT ILLNESS** had begun one month before admission with pain in the right side at the costal margin. The pain had come on after eating, was sharp, and radiated toward the back. The abdomen had become distended 10 days before admission and had been tapped and five liters of fluid removed. For four days he had been nauseated. He had remained conscious until late at night, two days before admission, and stupor had been present since that time. He had lost much weight during the past few months. The sclera had been yellow at times.

The **PHYSICAL EXAMINATION** showed the pupils to be small and fixed. The skin yellowish and bronzed in some places. There was cyanosis of the lips and ears. The teeth were greatly worn down. Respirations were infrequent. The lungs were negative but for a to and fro friction at about the 3rd intercostal space anteriorly over an area the size of a half dollar. The thorax was deep.

The abdomen was on the level of the ribs. The abdomen rose about 10 cm. above the level of the anterior superior spines. There was very slight bulging in the sides. The superficial vessels seemed very slightly distended and there were numerous enlarged venules. There were puncture marks midway between the umbilicus and the symphysis. The liver extended about 10 cm. below the costal margin; the edge was very sharp and hard. The surface was nodular in a few areas, and granular otherwise. There was slight oedema of the ankles. The scrotum was oedematous. There was a large distended vein from the left axilla, as a hepatic collateral.

The **LABORATORY EXAMINATIONS** showed: Urine dark brown in color and containing a trace of albumin and many hyaline and a few granular casts, and a few white blood cells. Bile reactions were strongly positive. **BLOOD:** Hb. 90 per cent; r. b. c. 4,576,000, w. b. c. 12,100. Differential count was normal. The Wassermann was negative.

Electrocardiograms showed left ventricular preponderance.

COURSE IN THE HOSPITAL. The T. P. R. chart showed Temperature 102, Pulse 110, Respiration 8 to 34. The patient gradually grew worse. Coma deepened and the patient died on the second day in hospital.

The **CLINICAL DIAGNOSES** were: Cirrhosis of the liver. Carcinoma of the liver, metastatic or primary, Ascites Arteriosclerosis, Hydrocele, Icterus, Fibrinous pericarditis.

The **PATHOLOGICAL DIAGNOSES** were: Hemolytic streptococcus septicaemia, Carcinoma of the liver with metastases to the lymph glands and the right adrenal. Haemochromatosis, Cirrhosis of the liver. Chronic inter and intralobular pancreatitis. Chronic passive congestion of the spleen and gastrointestinal tract. Ascites, Hydropericardium. Petechial epicardial hemorrhages. Fatty degeneration of the liver, kidneys and pancreas. Oedema of the lungs, chronic fibrous pleurisy. Chronic obliterative appendicitis. Hydrocele, Atheroma of the aorta.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS

KIDNEYS. The right kidney weighed 200 grams, the left 190 grams. The capsules stripped with ease, leaving a slightly granular surface. On section, the medulla and cortex were barely distinguishable. The cortex measured 5 mm. at its thinnest portion and its yellowish opaque linear markings were the prominent features.

Microscopically, there was a distinct atheromatous change in the vessels, but there was a loss of only a few glomeruli. The epithelial cells of most of the tubules of the cortex were filled with fat. The epithelium of an occasional tubule in the pyramids contained hemosiderin.

The **AORTA** was atheromatous, but still fairly elastic.

HEART. The heart weighed 350 grams. The epicardium at the base of the heart showed distinct punctate hemorrhages. The heart muscle was brownish in color. The cut surface of the auricle was of a light coffee color. The valves were normal except for moderate atheromatous degeneration, extending to the surface of the aortic cusp.

Microscopically, the amount of pigment granules at the ends of the nuclei of the cardiac muscle cells was perhaps somewhat greater than that in the average individual at this age. The ventricular muscle fibers showed advanced cloudy swelling in parts while in other parts cone shaped groups of yellowish brown pigment (hemofuscin) extended from the poles of the nuclei. There was a marked increase of fibrous connective tissue throughout the sections. The blood vessel walls were increased in thickness, and packed with red blood cells.

CASE 19—(51-75)

A man, aged 58, was admitted with the chief complaint of loss of voice, swelling in the throat, and difficulty in swallowing.

His **PAST HISTORY** was practically negative. When a child he had had considerable "lung trouble", but had had none the past few years. He had had herpes zoster 7 years previous to admission, and severe attacks of sinusitis a few years later.

His **PRESENT ILLNESS** had dated from a sudden onset, three months before admission, of difficulty in swallowing, which had gradually become worse. The thyroid cartilage had become prominent. A month later his voice had begun to get weak and he had become short of breath on talking or exerting himself. These symptoms had gradually increased, but the patient had continued to work until five days before admission to the hospital. At this time, he had become very dyspnoeic and had had to go to bed. Dysphagia and dyspnoea had rapidly increased, with progressive loss of voice and loss of about 15 pounds in weight. His appetite had been good, but he had been unable to swallow food. No pain and no vomiting had been noted.

The **PHYSICAL EXAMINATION** showed a slender, emancipated man, very weak and unable to speak above a whisper. There was very marked dyspnoea and a stridor on inspiration. An occasional hard cough was productive of mucopurulent sputum. The skin was warm and covered with perspiration and there was very marked cyanosis. The temperature was 103.8. A divergent squint of the right eye had been present since childhood. The mucous membranes were blue. No cranial nerve involvement was found. There was no tracheal tug. Just above the middle of the clavicle on the right was a hard mass, oval in shape, measuring 4x1 cm. and a similar mass was present on the left side.

The respirations were very labored, 32 per minute, with retraction of interspaces of interspaces on inspiration. The percussion note was resonant. The breath sounds were loud and high-pitched throughout the chest. In the left axilla and just below the left scapula, the breath sounds were harsher and occasional rales were heard. There was a retromammary dullness of 8 cm. in the first intercostal space and 6 cm. in the second intercostal space. No thrills or shocks were palpated. The cardiac dullness was not increased, measuring 2 cm. to the right, and 9 cm. to the left. The heart sounds were indistinct on account of the loud respiratory murmurs. No significant murmurs were heard; there was a questionable systolic murmur at the apex. The blood pressure was 110/78.

The larynx was free from any inflammatory reaction. The cords were absolutely immobile with a space between them of about 5 mm. There was a general thickening of the arteries.

The knee and Achilles and supinator reflexes on the right were slightly greater than those on the left. The abdominal and cremasteric reflexes were not obtained.

The **LABORATORY EXAMINATIONS** showed: Urine, 1.020 acid, sugar negative, with a trace of albumin and no casts, but occasional leucocytes. Blood: Hb. 90 per cent, R. B. C. 5,488,000, the W. B. C. 14,200. The Wassermann was negative. Sputum cultures showed B. influenzae predominating. The stools were negative.

Electrocardiograms were normal.

X-ray examination of the chest revealed a prominent arch of the aorta. A wide shadow extended up into the base of the neck, somewhat like the shadow of an enlarged manubrium. The left hilus shadow was very large. There was infiltration of the lower half of the left lung. Above, there were thickened lung markings with calcification. There were similar, but less marked, findings on the right side.

COURSE IN HOSPITAL: The breathing became more difficult, especially after slight exertion. The breath sounds increased in harshness gradually at both bases, with an increase in the rales. The sputum was mucopurulent and streaked with blood.

On the fourth day the condition showed a more rapid downward progress. The cyanosis was more pronounced. The respirations became more rapid and shallow. The pulse rose 168 per minute and was weak. The skin was cool and wet and dusky over the face, arms, and chest. The signs in the chest increased, involving the lower two-thirds of both lungs. The leucocytes increased to 18,700.

The patient died five days after admission.

The **CLINICAL DIAGNOSES** were: Bronchopneumonia (Bilateral) B. Influenza; vocal cord paralysis (bilateral); tracheal obstruction; carcinoma of the anterior superior mediastinum; arteriosclerosis (general); pulmonary tuberculosis (old healed); cervical adenitis.

The **PATHOLOGICAL DIAGNOSES** were: Carcinoma of the oesophagus with extension to larynx and metastases to the cervical lymph glands and liver; purulent bronchitis; bronchopneumonia; fibrinous pleurisy; caseous and calcified tuberculous nodules of both lungs and peribronchial regions and hilus; chronic diffuse nephritis; chronic prostatitis.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS

KIDNEYS: Both kidneys were of normal size. The capsules were slightly adherent. No striking changes were visible. Microscopically, the kidney tubules contained hyaline casts; many also contained granular debris. The glomeruli showed changes varying from thickening of Bowman's capsule to complete fibrosis of the glomeruli. Many contained a granular sediment. The fibrous interstitial tissue was definitely thickened in places and there were accumulations of lymphocytes in these areas. The pericardial sac was smooth and glistening throughout.

HEART: No gross abnormalities were evident. Microscopically, the muscle fibres were normal in size. A slight increase in connective tissue and a slight infiltration of leucocytes throughout the sections were noted. The endocardium was much thicker than normal and contained infiltrated leucocytes. The subepicardial fat was increased in amount and contained many infiltrated leucocytes.

CASE 20—(52-74)

A man, aged 68, was admitted with the chief complaint of pain in the back.

The **FAMILY HISTORY** was significant in that his father had died of "stomach trouble" and one sister of cancer of the stomach.

PAST HISTORY: He had used alcohol freely for years. In 1915 he vomited bloody material. He had had a chronic cough for many years. He had had an erythema on two occasions.

The **PRESENT ILLNESS** had begun insidiously with anorexia, which he noted during an attack of the erythematous skin condition. Off and on for months especially in the afternoon he had had attacks of dull aching pain in the small of the back. He also had had slight oedema of the ankles.

The **PHYSICAL EXAMINATION** showed a well developed, fairly well nourished man. The skin was pale and yellowish tinged, dry and scaly. There were large hard glands in the neck and the axillae. The lungs were emphysematous.

HEART: Systolic murmurs were heard over the mitral and aortic areas. The heart was enlarged slightly to left. The impulse of the apex was not visible or palpable. The heart sounds were distant. The aortic second sound was impure, but louder than the pulmonary second sound. The blood pressure was 158/68. The blood vessels were moderately thickened. **ABDOMEN:** There was a bulging between the ensiform and the umbilicus. No distinct pattern was made out. There was a visible mass in the right epigastrium. There was some slight dilatation superficial veins. The indefinite mass under the left costal margin and below moved with bimanual palpation. A round hard movable nodule was found in the skin over the right hypochondrium.

There was a marked oedema of the left leg from the hip down. The right ankle was also oedematous. The scrotum was oedematous. The prostate was small, soft and normal.

LABORATORY EXAMINATIONS revealed a hemoglobin of 51 per cent; red blood cell count 3,700,000; white blood cell count 8,400; Different count showed 33 per cent Lymphocytes; 9 per cent Endotheliocytes; 2 per cent Eosinophiles; 56 per cent Neutrophiles. The Wassermann was negative. The Tuberculosis Complement Fixation test was negative. The blood non-protein nitrogen was 36.5 mg. per 100 c.c.

The urine contained a trace of albumin, hyaline and granular casts. The specific gravity was rather fixed. Phthalein, when injected intravenously, was excreted to the extent of 15 per cent in 15 minutes, while, after subcutaneous injection, only 20 per cent was excreted in two hours. The stools gave a moderately strong positive guaiac test for occult blood. Electrocardiograms were essentially negative except for slight left ventricular preponderance. Gastro-intestinal fluoroscopic examination showed colonic motor delay and a spastic colon. Diffuse dilation of aortic arch was evident on an X-ray plate of the chest taken at seven feet.

Two days before death the patient vomited 50 c. c. of bloody matter.

The **CLINICAL DIAGNOSES** were: Carcinomatosis, Primary? Metastasis to skin. General adenopathy. Cirrhosis of liver? Oesophageal varices? Thrombosis of left saphenous vein. General arteriosclerosis. Hypertension. V. D. C. C. Mitral regurgitation. Aortic roughening (Arteriosclerosis). Simple anemia. Chronic intestinal nephritis.

The **PATHOLOGICAL DIAGNOSES** were: Carcinoma of stomach with metastases to the abdominal and thoracic lymph glands, to the liver, mucosa of the small intestines, pancreas, adrenal, skin, heart and wall of the left iliac vein. Hemorrhage from ulceration of primary growth in stomach and the metastases in the mucosa of the intestines. Thrombosis of left iliac vein. Chronic diffuse nephritis (small granular kidneys). Cirrhosis of liver (early). Interacinar pancreatitis. Chronic arteriosclerotic mitral and aortic endocarditis (slight). General anasarca and hydrops of serous cavities. Advanced arteriosclerosis. Hyperplasia of bone marrow.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS

KIDNEYS: The right kidney weighed 110 grams; the left 120 grams. They were quite similar in appearance. The fatty capsules were large. The fibrous capsules stripped leaving granular surfaces on which there were thin walled cysts. Vessels the size of a knitting needle entered the kidney substance from this capsule. On section the cortex and medulla could not be distinguished. The cortex presented opaque areas. The renal arteries were extensively calcified.

Microscopically, the normal markings were difficult to make out. Marked increase in fibrous tissue and many sclerotic glomeruli were noted. The arterial walls were greatly thickened and the vessel lumina completely obliterated.

The AORTA was sclerosed.

HEART: The pericardial cavity contained 200 c. c. of fluid. The heart weighed 420 grams. The myocardium contained numerous minute nodules scattered throughout. There were a few apparently fibrous thickenings of the epicardium and endocardium. Two of the aortic cusps were thickened to 4 mm. in places and showed some deformity. There was slight thickening and calcification of the mitral segments. The mitral valve measured 10.5 cm., the aortic 7.5 cm., the tricuspid 12.5 cm., and the pulmonary 9 cm.

Microscopically, the myocardium showed a slight increase in connective tissue throughout. In some areas there was a marked fatty atrophy of the heart muscle. The endocardium was slightly thickened. In the subendocardial fat there were numerous large and small metastatic nodules of adenocarcinoma. Some of these were very widespread and had a marked leucocytic infiltration in them. The myocardium below these metastatic nodules showed marked degenerative changes. Near the nodules there was a zone of simple necrosis which gradually changed to cloudy swelling as one proceeded away from the nodules. The smaller nodules were situated in definite relation to the blood vessels.

CASE 21—(26-100)

A girl, aged 17, was admitted with the complaints of shortness of breath and pain over the heart.

In her PAST HISTORY she had had attacks of acute rheumatic fever since the age of 9. The attacks had been worse in winter at which time the joints had become swollen, red, hot and very painful. She had had mild sore throats occasionally. She had been very nervous when young, but became more so between 8 and 9, when she had been unable to go to school because of "St. Vitus' Dance". The trouble improved in the summer only to start again when she tried to return to school. Chorea had been so severe that she had been unable to feed herself. The condition had continued until the age of 13, when it had stopped and had never returned.

The PRESENT ILLNESS had begun six months before admission with a severe attack of joint trouble, with much pain, swelling and fever. She had been in bed for two weeks, and convalescent for four weeks longer. She had returned to work and had felt well until four months before admission, when the symptoms had returned and persisted with pains in the knees, weakness, dyspnea and loss of appetite. Three days before admission she had had severe pains in the heart with fever and increased dyspnea which symptoms brought her to the hospital.

The PHYSICAL EXAMINATION showed: A pale, thin, cyanotic girl, slightly dyspnoic, coughing and perspiring. The cervical glands were enlarged but not tender.

The heart was enlarged, extending 12 cm. to the left and 4.5 cm. to the right. The apex impulse was diffuse. A blowing systolic murmur, and a questionable diastolic murmur were heard in the mitral area and at the aortic cartilage. A superficial "scratch" was heard over the lower sternum. There was tenderness over the lower sternum on pressure. There was a Corrigan pulse and a pistol shot sound in the femoral artery.

The abdomen was negative except for tenderness on palpation in the splenic region.

The LABORATORY EXAMINATIONS showed a hemoglobin of 54 per cent; R. B. C. 4,122,000; and W. B. C. 17,400. Blood cultures were negative. The blood pressure was: Arm 108/45, Leg 130/45. The urine was negative. The electrocardiogram showed prominent P waves and a tendency toward right ventricular preponderance.

COURSE IN THE HOSPITAL: Cough and dyspnea persisted with an irregular fever of 98° to 103° and a pulse rate of 120 per minute. Blood cultures were negative. The fever slowly subsided. The infected tonsils were removed. There was good operative recovery. Except for rises to 100° on two days the temperature remained normal. The pulse was 110. The pain and cough disappeared. The dyspnea persisted. The heart remained unchanged. The marked anemia persisted. Blood from a donor in Group IV (chronic nephritic) was secured for the patient who was in Group II. A transfusion of 400 cc. of citrated blood was given. A severe reaction ensued, with increasing cyanosis, a severe chill, and a fever of 103°. Slight relief was secured from adrenalin injections. A cough with a bloody sputum persisted. Dullness with distant bronchial breathing and rales was found in the right axilla and back. Diffuse petechiae and ecchymotic areas developed over both arms, the left side of the trunk, and the right buttock. Small septic skin lesions questionably embolic became generalized three days after transfusion. The symptoms and signs of heart failure progressed rapidly until the degree of oedema and congestion was extreme.

The liver became enlarged and tender, extending 6 cm. below the costal margin. The eyelids became swollen and the entire face puffy, and the ankles edematous. The urine showed a large amount of albumin and many hyaline and granular casts, white blood cells, and a few red blood cells.

She complained of pain in the lower precordium and in the splenic region. The spleen was not palpable. The enlarged liver was found to be pulsating. Auscultation showed a change in the murmurs. A loud diastolic was audible in the aortic area and was transmitted over the precordium. A tricuspid systolic and a questionable diastolic murmur differing from those heard at the mitral area were noted. Rough grating to and fro sounds were heard along the left border of the sternum. The blood cultures were negative. The urine

output was 100 cc. in 24 hours. The blood non-protein nitrogen was 47.6 mg. per 100 cc. Ecchymoses developed below both eyes. Both lung bases became dull. Rales were heard throughout both lungs. The hemoptysis persisted and the edema increased. The temperature was irregular and from subnormal 97 to 102°.

The patient died suddenly while on a bed pan. She had been in the hospital six and one-half weeks.

The CLINICAL DIAGNOSES were: Acute rheumatic endocarditis, chronic cardiac valvular disease, mitral stenosis and insufficiency and aortic insufficiency, cardiac failure with tricuspid regurgitation, cardiac hypertrophy and dilatation, pericarditis, pulmonary thromboses and infarcts, especially on the right.

The PATHOLOGICAL DIAGNOSES were: Chronic rheumatic endocarditis of the aortic, mitral, and tricuspid valves with acute warty vegetations, of the type characteristic of rheumatic endocarditis, cardiac hypertrophy and dilatation, pulmonary thromboses, and edema, atelectasis of the left lung, bilateral hydrothorax, ascites, anasarca, chronic passive congestion of the viscera, fatty degeneration of the liver, early aortic arteriosclerosis, uric acid infarcts in the kidneys. Cultures of the heart blood and the vegetations were sterile.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS

KIDNEYS: The right kidney weighed 140 grams, the left 110 grams. Both were purplish-red in color. The stellate veins were injected. The capsule stripped with slight difficulty. All cortical striations were distinct. There were a few golden-yellow deposits at the apices.

Microscopically, the walls of the tubules were edematous and granular. There was a granular sediment in the tubules and in Bowman's capsule. All capillaries and larger vessels were much congested. The glomerular capillaries were markedly congested.

AORTA: There were a few white nodular thickenings near the aortic valve region and a number of yellowish-white streaks running lengthwise along the aorta. A few nodular thickenings were noticed at the origins of the thoracic branches. Microscopically, there was a slight thickening of the intima.

HEART: The pericardial cavity contained 75 cc. of straw-colored fluid. The epicardium was slightly thickened and a few adhesions were present. The heart looked quite large as it lay in position. It was definitely dilated and hypertrophied and weighed 235 grams. The hydrostatic tests showed definite mitral and tricuspid insufficiency. The aortic valve measured 6 cm. in circumference. At the edges of the valve were a number of minute warty vegetations, most of which appeared old. Near the border of the cusp there was a bead-like arrangement. The largest nodule was 3 mm. in diameter. The valve leaflets were thickened and slightly contracted. The mitral valve measured 10 cm. The edges of the leaflets were thickened and contained a large number of minute sessile vegetations of warty appearance. The pulmonary valve measured 7 cm. and appeared normal. The tricuspid valve measured 12 cm. The free borders of the flaps showed a chain of vegetations, the largest of which was 4 mm. in diameter and extended straight upward from the valve. This nodule was a yellow-gray color. The vegetations were slightly blood-stained and, for the most part, looked old, although some appeared fresh.

Microscopically, the valves all showed chronic fibrosis. Some vegetations contained fibrin deposits, but no bacteria, and most of them were old organized lesions.

The subepicardial myocardium showed a marked degree of degeneration. The muscle fibres showed some diffusion of chromatin and some showed a simple necrosis with complete destruction of the fibres. Some of the fibres showed a fatty degenerative infiltration also. There was no change in the endocardium, but the epicardium showed some thickening. The majority of the muscle fibres showed some degree of cloudy swelling. In some areas there was a slight mononuclear, leucocytic infiltration. There was only a slight increase in the connective tissue.

CASE 22—(13-13)

An unmarried woman, aged 70, was admitted with the complaint of a lump in the left breast and diabetes.

The FAMILY HISTORY was unimportant, except that one sister had been operated upon 4 years previously for cancer of the left breast. There had been no recurrence. There was no other case of diabetes in the family.

The PAST HISTORY: She had had the usual childhood diseases; malaria at 23, articular rheumatism for 20 years and occasional tonsillitis. There was no history of cardiac or pulmonary symptoms. Four years before admission, she had lost considerable weight and had been told that she had diabetes. There had been severe itching. No urinary symptoms were noted other than nocturia once. She had noticed a gradual development of lumps in the fingers and toes for twenty years, which she had associated with her rheumatism. She had had no neuro-muscular symptoms. Her menstrual history was negative, with her menopause at 40 years. She had never been married and had had no pregnancies. Her average weight had been 165 pounds up to four years before admission. Six months previous to admission she weighed 136 pounds, while at admission her weight was 121 pounds. She had eaten no sugar for the last year, otherwise her diet had not been definitely prescribed.

Her PRESENT ILLNESS had begun six months before admission with pain in the left breast above the nipple. The lump had been noticed at that time and had progressively enlarged. One month before admission the lump had become ulcerated. There had been a progressive loss of weight and strength. Two days before admission, a sore spot had developed in the left foot.

The PHYSICAL EXAMINATION showed a moderately well nourished and well developed woman. The left pupil was smaller than the right and slightly irregular; otherwise they were negative. The neck showed no glandular enlargement. The pulsations in the veins were noticeably forcible. There was no thyroid enlargement. The chest was slightly barrel-shaped. The lungs were negative to percussion. A few moist rales were heard at

the bases. The heart presented no shock and no thrills. The apical impulse was diffuse in the 5th intercostal space 12 cm. to the left. A rough systolic murmur was heard at the aortic cartilage and at the apex, whence it was transmitted to the axilla. No diastolic murmurs were heard. The second sound was sharp. A capillary pulse was present. Moderate thickening of the arteries was noted. The blood pressure was 150/58 and 172/60.

In the upper half of the left breast there was a mass the size of half a baseball. The surface presented an ulcer with a dirty gray granular base. The mass was hard, attached to the skin everywhere over its anterior surface and rather nodular. The mass moved readily with the breast over the chest wall. The axillary glands on the left were palpable, but not hard. They were slightly larger than the glands on the right. The abdomen was negative except for an umbilical hernia. The extremities showed Heberden's nodes on both fingers and toes and bilateral Hallux valgus. There was a small dry ulcer on the left middle toe. The rectal examination was negative.

The LABORATORY EXAMINATIONS showed: hemoglobin 90 per cent, R. B. C. 4,136,000, and W. B. C. 26,000. The differential count was: polymorphonuclears 85 per cent, eosinophiles 1 per cent, transitionals 1 per cent, lymphocytes 9 per cent. The urine was acid, and contained sugar and albumin, and no casts, a few W. B. C. and R. B. C.

COURSE IN THE HOSPITAL: She was given a diet containing 100 to 200 grams of carbohydrate. She was hypercsthetic over the legs and feet, probably because of an early neuritis. The blood CO₂ volume per cent was 57.45, the non-protein nitrogen was 33 mg. per 100 cc, the blood sugar on admission was 0.28 per cent, later 0.2 per cent. She was given 40 grams of carbohydrates as green vegetables. The kidney function test with 1 cc. 'phthalein given intramuscularly showed in 3 hours an output of 84 per cent. On another examination the urine was acid, there was no sugar, no albumin, and only a few W. B. C. and R. B. C. were present.

A radical breast amputation was done, beginning with Rodman's incision on the left side, then followed a radical dissection of the axilla, removing the sternal portion of the pectoralis major and the pectoralis minor, together with the breast. Complete closure was made with a stab drain through the posterior axillary wall. The patient took the anesthetic well and was in good condition throughout the operation, which lasted 1½ hours.

At the dressing the skin flaps were noted to have a satisfactory circulation. The patient was in good condition. The urine, however, showed much sugar and diacetic acid. The CO₂ volume per cent was 49.3. Fluids were forced and sodium bicarbonate was given by mouth. The pulse rate was 120. The electrocardiograms showed auricular fibrillation. 10 cc. of digitalis were given in divided doses. Sodium bicarbonate was given intravenously. The respirations were rapid. There were rales and distinct tubular breath sounds, with increased whisper and a slightly dulled percussion note in the bases.

The patient became much worse. She was irrational. The temperature was 101°, pulse 140, respiration 36. The urine showed much sugar and diacetic acid and a trace of albumin, W. B. C., and granular and hyaline casts. The leucocytes rose to 16,500. There was probably a high pulse deficit, but this could not be determined on account of the wound over the cardiac region. She died eleven days after admission.

The CLINICAL DIAGNOSES were: Diabetes mellitus, carcinoma of the left mammary gland, arteriosclerosis, chronic cardiac valvular disease, mitral insufficiency, chronic infectious arthritis, papilloma of the cervix, suppurative pleurisy, chronic interstitial nephritis, auricular fibrillation, cardiac failure, acidosis.

The PATHOLOGICAL DIAGNOSES were: General arteriosclerosis, chronic interstitial nephritis, sero-purulent left pleurisy and pericarditis, cardiac hypertrophy, atrophy of the pancreas, atrophy of the ribs and sternum, cloudy swelling of the viscera, hyperplasia of Peyer's patches and the solitary follicles, wound of the radical removal of the breast for carcinoma, focal fibrous nodules in the lungs and peribronchial lymph glands, scar in the right lung apex, chronic localized fibrous pleurisy on the right, chronic perisplenitis, multiple simple cysts of the liver and kidney, atrophy of the appendix and genitals, chronic localized pericarditis, umbilical hernia, papilloma of the cervix, hemorrhoids. Blood pericardial, pleural, and lung cultures showed streptococcus hemolyticus.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS

KIDNEYS: The right kidney weighed 170 grams and measured 12x6x3.5 cm. The left kidney weighed 190 grams and measured 13x7x3 cm. The capsules stripped with some resistance and the exposed surface had a finely pebbled appearance. Just beneath the capsule of each kidney were a few greenish areas which on section had caseous centers. On section cloudy swelling, and but little if any fatty degeneration was present. The line between the cortex and the medulla was indistinct. Both cortices were somewhat thin, measuring 8 mm. There were a few small cysts in the substance of the right kidney.

Microscopically, the kidneys showed rather widely separated abnormal areas of connective tissue or scar, with round cell infiltration. There was also round cell infiltration about the glomeruli and tubules. The tubules contained casts. The parenchyma was swollen and granular. The vessel walls were thickened. Section through a cyst showed a narrow lining of connective tissue beneath which the tubules were compressed and the cells flat. At one point a few low columnar cells, possibly cyst epithelium, were seen.

The AORTA showed massive atheromatous plaques with a few ulcerations and calcified areas. The coronary vessels were tortuous and hard.

The HEART weighed 330 grams. The aortic valves were uniformly slightly thickened. There was calcification in small areas of the aortic ring. The auricles showed nothing of interest. The foramen ovale was open, but covered with a valve-like leaflet. The valves after fixation measured as follows: aortic 6.5 cm., mitral 7.5 cm., tricuspid 9.5 cm. The mitral valve was uniformly thickened. A mass on the septal side of the ring contained caseous and calcified material. In other places the central portions of the thickened ring were filled with yellow coarsely granular thick material. The thickened calcified areas of the mitral ring extended well into the myocardium. Externally there were no evidences of myocarditis. The epicardium contained a moderate amount of fat.

Microscopically, there was a marked fatty atrophy throughout the sections. One of the sections showed a large area of calcification, along the border of which there was a marked leucocytic infiltration. The epicardial fat was increased, but the epicardium was also slight-

ly thickened. The endocardium showed no marked changes. Some of the blood vessels showed a marked acute congestion.

CASE 23—(44-82)

A married woman, aged 41, was referred to the hospital by her physician because of pain in the lower abdomen with vomiting, for three days.

The PRESENT ILLNESS had had a rather sudden onset, six days before admission. The pain was said to have begun with aching in the lower abdomen which gradually increased until on entrance it was sharp and very severe. The bowels had moved daily. She had menstruated a month previously and then again in small amount during the last few days. She denied any attempt at abortion. She had had no previous attack similar to this one.

On PHYSICAL EXAMINATION the patient was found lying quietly in bed, not complaining of pain, but stating that movements involving her abdomen caused severe pain. The abdomen was distended moderately. There was no visible peristalsis. On palpation the entire lower abdomen below the umbilicus was very tender on pressure and there was rigidity of both sides of the abdomen. The upper abdomen was only moderately tender and also rigid. The liver was not palpable and not tender. There was no marked epigastric tenderness. No evidence of free fluid was obtained. There was a vaginal discharge with a rather marked odor and sero-sanguinous in nature. Vaginal examination showed a firm cervix, second degree retroversion of the uterus, which was about 50 per cent enlarged, movable, and slightly softer than normal. There was no bulging of the cul de sac. No right pelvic mass was felt, but a slight sense of fullness in the left pelvis was noted. Tenderness elicited on vaginal examination was less than that on abdominal examination. Rectal examination revealed nothing noteworthy.

A scar was present on the right thorax caused by a radical removal of the breast for carcinoma seven years previously. The right arm above the elbow was somewhat swollen and red. The patient stated that this condition of the arm had followed an infection on the side of the hand about a week before admission. The lesion on the hand was healed.

Examination of the heart and lungs showed nothing unusual. The temperature was 103° pulse 124, and respiration 26. Four hours after admission the patient had a chill and her temperature rose to 106°, with pulse to 145 and respiration 30. The leucocyte count was 21,600.

COURSE IN HOSPITAL: On the morning of the second day, the patient's condition was about the same. Laparotomy was performed through a vertical incision just to the right of the midline and just above the symphysis. About 100 cc. of turbid fluid escaped, containing many flakes of fibrin both large and small. No abnormal odor was noted. The peritoneum was everywhere hyperemic. There was numerous fibrinous deposits on the intestines. No masses were felt anywhere. The uterus was slightly enlarged and soft, moderately retroflexed and freely movable. The tubes and ovaries were not enlarged and not adherent. The appendix was about 4 inches long and apparently not more inflamed than other organs. Appendectomy was performed in the usual way. A considerable portion of the small intestines and sigmoid was searched for diverticula, but none were found. The upper abdomen was not satisfactorily explored.

Following the operation the patient showed no improvement. The temperature fluctuated between 101° and 104.5°. The pulse was always rapid and approximately 130, until the day before death, when it reached 170 per minute.

On the fourth day after the operation coarse bubbles were heard all over both lungs. There were no signs of infiltration in front. The patient was too ill to permit satisfactory examination posteriorly. The blood cultures made on the second day were sterile. A culture of the peritoneal fluid made at operation showed streptococcus pyogenes and staphylococcus aureus.

LABORATORY FINDINGS: The urine on admission showed a trace of albumin. No sugar was found. The reaction was acid and the specific gravity was 1.025. There were many granular casts. The urine on the fourth day showed a large amount of albumin and many hyaline and granular casts. Electrocardiogram showed no preponderance. The patient died, apparently from the toxemia of the sepsis, on the seventh day after admission.

The CLINICAL DIAGNOSES were: Acute general peritonitis, acute axillary lymphadenitis, cellulitis of the right arm, acute appendicitis, septicemia?, bronchopneumonia, edema of the lungs, acute fibrinous pleurisy, retroversion, chronic endometritis, chronic vaginitis and ileus.

The PATHOLOGICAL DIAGNOSES were: Fibrino-purulent peritonitis, slight bilateral bronchopneumonia, purulent bronchitis, acute splenic tumor with hyaline necrosis of the splenic nodules, appendectomy, operative removal of the right breast and axillary nodes with the old scar extending into the axilla, retention cyst of the pancreas, cholelithiasis, generalized hemolytic streptococcus infection.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS.

KIDNEYS: The kidneys were similar in appearance and together they weighed 420 grams. The capsule stripped with ease, leaving a surface which was slightly granular in places. The cut surface projected above the cut capsular surface.

Microscopically, many glomeruli appeared large and had the appearance of having an increased number of nuclei. In places, the cells lining the capsule spaces were cuboidal or even columnar, but there was no piling up. There were a few leucocytes within the capsular spaces, the capillaries contained great numbers of neutrophilic polymorphonuclears. The epithelium of the convoluted tubules was swollen and in the cells there were fine vacuoles. The tubules contained albumin, granular and occasionally hyaline casts.

HEART: The heart weighed 280 grams and was negative externally. No vegetations could be seen on any of the valve leaflets. There were no evidences of a fibrosis or old healed valvulitis. The valvular orifices were normal in size.

Microscopically, there was an advanced fatty atrophy throughout the myocardium, especially in the tissue immediately beneath the subpericardial fat. There were hemorrhagic areas in the subpericardial fat. There was also a slight leucocytic infiltration throughout

the myocardium. Some of the muscles showed an advanced cloudy swelling. In some areas, especially in the auricular muscle there were spherical and oval vacuoles varying in size from very minute spaces to ones the size of the nucleus.

CASE 24—(29-97)

A hostler, aged 71, was admitted with the complaint of shortness of breath, swelling of the legs and abdomen.

The FAMILY HISTORY was significant in that his mother had died of heart trouble. His father had died of apoplexy and a brother of Bright's disease.

In his PAST HISTORY he had had scarlet fever, smallpox, and asthma, gonorrhea at 20, colic with jaundice at 20, acute rheumatic fever at 25. There had been no complications. For some time he had had to urinate 8 to 12 times a day and 5 to 6 times at night.

His PRESENT ILLNESS had begun one year before admission with a "dizzy spell", which had been followed by a gradually increasing dyspnea and palpitation, chronic cough and choking spells. The extremities had been swollen for a few weeks, five months before admission. Edema had reappeared three weeks previous to admission and had persisted. Orthopnoea had been present for one month.

The PHYSICAL EXAMINATION showed dyspnea, orthopnoea, with slight icterus and cyanosis. There was marked edema over the back and legs. The neck veins were engorged and an auricular form of venous pulsation was present. The lung bases were dull with distant breath sounds, egophony, and rales.

HEART: The cardiac impulse was just palpable in the 6th intercostal space 17 cm. to the left of the midsternal line. The right border was 4 cm. to the right of the midsternal line. The heart sounds were distant. The pulmonary second sound was louder than the aortic second sound. No murmurs were heard. Many extra systoles were present, but the pulse was otherwise regular. The blood pressure was 150/100. The blood vessels were thickened and tortuous. The abdomen was distended in the flanks and a shifting dullness was present. The liver was 4 cm. below the costal margin, smooth and not tender. The prostate was enlarged. The genitalia and extremities were edematous.

The LABORATORY FINDINGS: The urine contained a heavy cloud of albumin; the sediment showed hyaline and granular casts, w. b. c., and occasional r. b. c. The specific gravity ranged between 1.019 and 1.025. The night volume was less than the day volume. The pththalein excretion was 40 per cent in 2 hours. The blood: Hemoglobin 70 per cent; R. B. C. 3,530,000; W. B. C. 6,200. The Wassermann reaction was negative. The blood non-protein nitrogen was 44.4 mg. per 100 cc. The vital capacity was 1700 cc. to 2000 cc. Electrocardiograms revealed a slight left ventricular preponderance, with left and right ventricular extrasystoles.

COURSE IN HOSPITAL: With rest for 5 days, he lost 2.2 kilos in weight. Theocin was ineffectual. Digitalis tincture in a massive dose of 19 cc. was given. Pulsus alternans was noticed 1½ hours later, and tachycardia, rate 160, 3½ hours after administration. The patient was doing very poorly and the digitalis and other cardiac stimulants were of no avail. The patient died five hours after the digitalis dose, just one week after admission.

The CLINICAL DIAGNOSES were: Chronic myocarditis, cardiac hypertrophy and dilatation, general arteriosclerosis, bilateral hydrothorax, hydropericardium, ascites, anasarca, chronic interstitial nephritis, chronic passive congestion of the liver, hypertrophy of the prostate.

The PATHOLOGICAL DIAGNOSES were: Cardiac hypertrophy and dilatation, chronic passive congestion of the liver, spleen, and kidneys. Subpleural and subepicardial hemorrhages. Hydrothorax, hydropericardium, ascites. Chronic diffuse nephritis in a contracted red kidney; fatty degeneration of the liver; atrophy of the testicles, hypertrophy of the prostate with calculi. Healed pulmonary tuberculosis with old pleural adhesions, anthracosis. Jejunal diverticuli.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS.

KIDNEYS: The fatty capsules were abundant. The right kidney weighed 150 grams and measured 11x6.5x3.5 cm.; the left weighed 165 grams and measured 12x5.5x3 cm. The capsule was somewhat adherent and on removal it left a somewhat granular surface of deep purplish-red color. There were numerous small cysts measuring 1 to 3 mm. On section the cortex averaged 4 mm.; at its narrowest point it was 2.5 mm. in thickness. The cortical striations were seen to be somewhat irregular. The arteries were all somewhat prominent and stood open and the larger ones were thickened.

Microscopically, there was intense congestion of all the vessels, those of the tubules as well as those of the glomeruli. The larger vessels showed distinct thickening, especially of the intima. There were areas of connective tissue infiltration with total occlusion of the tubules in many instances. The epithelium of the tubules was, for the most part, flat and the lumina were filled with a loose granular, pink-staining debris. An occasional tubule was seen in which the epithelium appeared swollen and granular.

The AORTA was somewhat elastic. There were a number of yellow nodules over the intimal surface, but these changes were not so conspicuous as they were in the smaller vessels.

Microscopically, the intima showed only a small amount of thickening with a few areas in which a few round cells were seen. The media and adventitia showed no changes.

The HEART extended from the right border of the sternum to the left anterior axillary line. The right side of the heart was very much distended with blood. The heart muscle mass weighed 600 grams. The valves were all competent by the hydrostatic tests. The valve rings measured as follows: aortic 8 cm., pulmonary 6.5 cm., tricuspid 7.5 cm., and mitral 8 cm. Along the descending branch of the left coronary artery, there were a number of echymotic spots 1 to 2 mm. in diameter. These were distributed over an area of about 3x2 cm., which extended from the bifurcation of the descending branch of the left coronary to the second branch. A few subepicardial hemorrhages were seen over the left ventricle toward the apex and over the right ventricle. The coronary vessels had numer-

ous yellow patches on their intimal surfaces and their walls were thickened. A small branch of the left coronary was seen to go to the left auricle.

Microscopically, there was a conspicuous increase in connective tissue throughout the sections. The epicardium was thickened and showed an advanced necrosis beneath which the myocardium also showed a marked necrosis. The cell outlines could not be distinguished. The blood vessels of the myocardium were markedly increased in thickness and at the same time gorged with red blood cells. The endocardium was thickened.

CASE 25—(18-175)

A white man, aged 63, was sent in from the O. P. D., complaining of asthma-like shortness of breath and swelling of the lower half of the body.

The FAMILY HISTORY was somewhat significant in that his mother had died of heart disease at 76, two brothers of chronic alcoholism, and one sister of obesity and mental deterioration.

He had been married for 42 years. His wife was living but had been in poor health, suffering from chronic arthritis and very poor vision. She had had one miscarriage early in life and had had no other pregnancies, the reason for which was not known.

He had been a house painter for 47 years.

He had always been a heavy meat eater. He had used alcoholic drinks in moderation except for considerable beer and whiskey for one or two years.

In his PAST HISTORY he had had no infectious disease, except for one attack of gonorrhea. He denied syphilis. Eight years before admission he had had "blood poisoning" in the right hand and arm, which was successfully treated by incision and injection of a sulphur-like solution. He had suffered from insomnia and nocturia.

His PRESENT ILLNESS had had its onset five months before admission when, through exposure to cold and dampness and overwork, he had contracted a severe chest cold, probably an extensive bronchitis. At this time he had noted shortness of breath and occasional sharp pain over the heart. He had done only week's work after the onset of the cold. For one month previous to admission he had been unable to work at all. For three weeks he had had orthopnea, and "smothering" nocturnal dyspnea. Five days before entering, his ankles had begun to swell, the swelling extending up the leg very rapidly (in fact overnight). Two days later a redness had spread over the legs, penis, and scrotum. The face, abdomen, and hands had not swollen.

The PHYSICAL EXAMINATION showed orthopnea and an expression of anxiety. The face was flushed and showed distended venules over the cheeks. Four purpuric spots were noted over the back of the left hand, and extensive subcutaneous hemorrhage was present over the legs. There was a massive edema of the feet, legs up to the hips, and the scrotum. Eyes: a mild bilateral albuminuric retinitis was noted; the pupils were unequal and reacted very sluggishly to light. The ears and nose were negative. The lips were slightly cyanotic. Very severe pyorrhea, and many carious teeth were present. The tonsils were apparently not abnormal. The neck veins were markedly engorged. No palpable glands were present. The thyroid gland was negative.

The chest was deep. There was retraction of bases on inspiration. The percussion note was impaired at the bases. A few coarse rales were heard at the bases. Cheyne-Stokes respirations were noted.

The heart was greatly enlarged, the dullness extended 6 cm. to the right and 14 cm. to the left of the midsternal line. The cardiac impulse was not seen or felt. The heart sounds were loud at the apex where there was a protodiastolic gallop rhythm and a sharp systolic murmur, which was well transmitted to the axilla and toward the midline. The aortic second sound was distant but ringing. Occasional extrasystoles were noted. The blood pressure was 200/120 and 185/125.

The radial, and brachial arteries were palpable, hard, thickened and tortuous. The pulse was regular with, however, alteration.

The abdomen was above the costal margin. The panniculus was thick. Some dullness was present in the flanks, but it was not definitely shifting and was without a definite fluid wave. The liver was felt 6 cm. below the rib borders, but was not distinctly tender.

The reflexes were present and active. The prostate was slightly enlarged. The right testicle was larger than the left.

The LABORATORY EXAMINATIONS showed: Urine, acid, with a specific gravity of 1.012 to 1.030, and containing 3 to 8 grams of albumin per liter, hyaline and granular casts. The twelve-hour urine output exceeded the intake except on the last day. 36 per cent on the phthalein injected was excreted in two hours. The blood showed: 70 per cent Hgb., R. B. C. 4,720,000, W. B. C. 9,000; the differential count was normal. The Wassermann reaction was negative. The non-protein nitrogen was 45 mg. and the uric acid was 5.21 mg. per 100 cc. of blood. The CO₂ volume per cent was 51. The X-ray plate of the heart taken at 7 feet showed M. R. 7.5 cm., M. L. 11 cm., L. 18 cm., and A. 6.5 cm.

The electrocardiograms showed evidence of myocardial changes, left ventricular preponderance. The P-R interval increased to .24-.25 sec. with partial block, which was variable and later of high degree, complete in parts, with sinus arrhythmia and frequent extrasystoles, and finally, auricular fibrillation.

COURSE IN HOSPITAL: The edema and orthopnea increased gradually. 15 cc. Tincture of Digitalis was given and some improvement was noted. Seven days after admission the patient became irrational, overpowered the nurse, and fell downstairs. He showed no ill effects, but the next morning he was found dead in bed.

THE CLINICAL DIAGNOSES were: Chronic myocarditis, partial heart block, auricular fibrillation, cardiac hypertrophy and dilatation, chronic nephritis, and albuminuric retinitis.

The PATHOLOGICAL DIAGNOSES were: Chronic mitral and tricuspid endocarditis with calcification; cardiac hypertrophy; chronic diffuse nephritis; general anasarca; chronic passive congestion of the viscera, including the spleen, which was enlarged; chronic fibrous epicarditis; pleurisy and perisplenitis; lipomatosis of the pancreas.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS:

KIDNEYS: The right kidney weighed 150 grams and measured 10.5x6.5x2.5 cm., the left weighed 170 grams and measured 11x6x3 cm. The cortex of the right measured 5 mm. in thickness, while that of the left measured 7 mm. The capsule stripped without difficulty, in each case exposing a deep, rough, granular surface upon which several cysts were seen in each kidney. The cut surfaces were also deep red in color. The line of demarcation between the cortex and medulla was irregular and often obliterated by scars. The glomeruli could be discerned as yellowish-gray foci, nearly one millimeter in diameter. Many of the tubules appeared as yellowish-gray strings alternating with intervening deep red lines.

Microscopically the kidneys showed large scars and numerous tubules filled with hyaline casts. Many of the scars were rather heavily infiltrated with small round cells. Cloudy swelling of the tubular epithelium was advanced.

The **AORTA** on the whole, showed very little change; only a few rather small fatty plaques were seen.

The **HEART** was greatly enlarged and weighed 700 grams. The epicardium over the ventral aspect was rose red in color, due to the presence of innumerable petechial hemorrhages into it. The petechial hemorrhages were scattered elsewhere in the epicardium, but were less numerous. The aortic valve appeared normal. There was calcification around the base of the mitral valve and also, but to a less extent around the tricuspid valve ring.

The calcium deposit was almost uniformly distributed around the base of the mitral valve. Calcification was also taking place in some of the papillary muscles on the left side. The pulmonary valve appeared normal. Both the mitral and tricuspid valves appeared incompetent with the hydrostatic test.

Microscopically, in one portion of the myocardium, there was an enormous collection of polymorphonuclear leucocytes. In this area much of the muscle tissue had been destroyed and only parts of cells remained. There were some other indications of foci or grouping of the leucocytes in the myocardium, indicating small abscesses. The subepicardial tissue showed some hemorrhage and the myocardium showed simple necrosis in places, while in others there was advanced cloudy swelling. There was a conspicuous increase in connective tissue throughout the sections. The endocardium was thickened.

CASE 26—(8-8)

A white boy, aged 17, was admitted with the complaint of tenderness in both legs, pain in the throat, and drowsiness.

The **FAMILY HISTORY** and **PAST HISTORY** were irrelevant.

The **PRESENT ILLNESS** had begun as an acute infection. Previous to one month before admission he had been seemingly in perfect health. He had first suffered from vomiting and constipation. These symptoms had been followed, after several days, by fever, which at one time had reached 103° F. At the time of the fever and following it, the patient noted difficulty in standing when his eyes were closed. He had tended, at such times, to fall always to the right. During this same time and even for some time previously, he had suffered from transient attacks of diplopia. An examination by an eminent neurological surgeon had shown a subnormal temperature, nystagmus, hyperanemia and edema of both optic disks, general muscular weakness, and slight muscular atrophy, especially in the lower extremities. The Babinski's and Chaddock's reflexes were positive on the left and, possibly, also on the right. The tendon reflexes were brisk to exaggeration throughout, particularly in the left lower extremity. His speech was slurred and inarticulate. Nystagmus was marked when the patient looked to the right, left, and upward. The sense of motion and position was poor and there was marked ataxia elicited by the tests. The cerebrospinal fluid was found to be negative. The leucocyte count was 16,000.

The patient had begun to complain of numbness and tingling sensations, at first in his left foot and leg and then in the right lower extremity. The deep reflexes were absent at the ankle and very inactive at the knee, and plantar irritation gave no response. Hyperesthesia was present on the left side and similarly on the right well up to the second intercostal space. The changes in the optic disc persisted. The paralysis of the lower extremity had increased so that he was unable to flex his foot. The nerve trunks had become tender and Lasegue's sign was positive.

One month after the onset of his symptoms, he had begun to complain of tenderness in both legs and pain in the throat, also drowsiness and drooping of the eyelids.

The **PHYSICAL EXAMINATION** revealed slight changes from those previously noted. The fundus picture suggested a receding optic neuritis. The breathing was shallow and slow. The left leg was completely paralyzed, the right could be flexed at the knee with difficulty. The knee kicks were absent, while the Babinski's sign was positive on each side.

The heart was not enlarged and was negative except for a systolic murmur at the apex. The lungs were negative.

The **LABORATORY FINDINGS** were of no great significance. The urine varied in specific gravity from 1.013 to 1.026. Traces of albumin were present at times.

The blood examinations showed 70 per cent hemoglobin; 4,000,000 red blood cells; and 12,000 to 8,000 white blood cells. The blood Wassermann was negative. The spinal fluid contained 6 lymphocytes per cu. mm. and showed a positive Pandy reaction, but a negative Wassermann, a 5554320000 colloidal gold reaction, and negative smears, culture, and animal inoculations.

The electrocardiograms suggested a left ventricular preponderance.

COURSE IN THE HOSPITAL: Tube feeding had to be resorted to and spontaneous vomiting occurred during the day. The patient gradually lost ground. The delirium and lethargy increased. The pulse became rapid. The temperature rose above 101° only during the last few days of his life and reached 108.5° and the leucocytes rose to 19,400 with the terminal pneumonia. He died just two months after his admission; that is, exactly three months from the onset of his symptoms.

The CLINICAL DIAGNOSES were: Acute lethargic encephalitis, myelitis, neuritis, optic neuritis, and terminal pneumonia.

The PATHOLOGICAL DIAGNOSES were: Acute polioencephalitis inferior, acute myelitis, widespread meningeal inflammation, neuritis of the optic, 3rd, 4th, 5th, 6th, 7th, and 8th cranial nerves, acute cerebral edema, chronic passive congestion of the viscera with cloudy swelling, broncho-pneumonia, acute congestion and hemorrhage into the right conjunctiva and duodenum, chronic prostatic urethritis, fatty changes in the liver.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS:

KIDNEYS: The right kidney weighed 110 grams and measured 10.2x6x3 cm. The left weighed 120 grams and measured 10.5x5.7x3.75 cm. The capsules stripped easily, revealing slight evidence of foetal lobulation. The stellate veins were congested. The cortex had a grayish-red color and measured 6 mm. in thickness. The medulla was congested. The interlobular vessels of the cortex were filled with blood, which made the gray columns stand out prominently. Microscopically, the parenchymal cells lining the secreting tubules were swollen and granular.

The AORTA was elastic and the intima was smooth except for a few linear fatty plaques near the base.

The HEART weighed 210 grams. The epicardium contained very little fat. The valves appeared normal except that the mitral and tricuspid rings seemed dilated to a slight extent.

Microscopically, there was a diffuse leucocytic, as well as fibroblastic, infiltration throughout the myocardium. The epicardium showed marked degenerative changes beneath which areas the myocardium showed an advanced simple necrosis. There was also a very marked fatty atrophy of the epicardial myocardium at various places throughout the heart. The subepicardial fat seemed abundant. The blood vessels showed thickening and congestion. The endocardium was only slightly thickened.

CASE 27—(52-72)

A married woman, aged 56, was sent in from the O. P. D. complaining of nausea and vomiting after every meal, pain in the stomach, loss of weight and weakness.

The FAMILY HISTORY was irrelevant. She had been married at 22. Her husband had died six years previously in an accident. She had been pregnant 17 times, and had miscarried the fourth, seventh, tenth and eleventh times. Seven children had died in infancy. Six children were living and well.

In her PAST HISTORY she had had "chills" at 12 for one year, typhoid at 23, tonsillitis and sore throat frequently to 23, sciatica at 43. She had had rheumatism at 45, at which time both feet were painful and swollen, and she had remained in bed for several months. She had been jaundiced slightly at this time.

Palpitation had been noted on exertion and during excitement for many years.

Her PRESENT ILLNESS had been dated from a fall down a flight of 16 stairs 7 months previous to admission. She had been unconscious for a while, then she had begun to retch and vomit. She had vomited frequently after this without relation to meals. The vomitus had consisted of "frothy material." No food could be retained. She had been jaundiced for about two weeks. No pain had been noted at first. Six months before admission she had begun to have "gripping" pains in the mid-epigastrium, which spread to either side posteriorly. These had not been constant, but would come and go, sometimes they had been absent for several days. Pains had been worse after meals especially after heavy ones, coming on in 15 to 30 minutes, and remaining unrelieved by food. The vomitus had come up in mouthfuls soon after eating. Her vomitus had occasionally been blood streaked. She had lost ambition, appetite, strength and weight. Her best weight had been 145. Her weight on admission was 78. She had had to quit work six weeks before admission because of weakness and a gnawing epigastric pain.

The PHYSICAL EXAMINATION showed extreme emaciation, some cachexia, and an anxious expression. Her pupils were very slightly irregular and unequal, but reacted to light and accommodation. The mucous membranes were slightly cyanotic. Most of the teeth were missing. The gum margins were eroded, pus was expressible. The tonsils were negative.

No abnormal pulsations were noted in the neck.

The lungs were clear; no rales were heard.

HEART: The cardiac impulse was diffuse, extending over the precordium. The point of maximum intensity was in the fifth intercostal space, 8 cm. from the midsternal line. The heart sounds were regular and of fair quality. M2 was muffled, M1 strong, and P2 was greater than A2. The radials were thickened. The pulses were synchronous, of good volume and tension. The brachials were thickened and tortuous. The cardiac girth extended 4 cm. to the right and 10 cm. to the left of the midsternal line. No murmurs were heard. The blood pressure was 140/104.

The Abdomen was scaphoid with a prominence in the epigastrium, especially to the left. No peristalsis was visible. There was a systolic impulse in the epigastrium. A mass moved down with deep pressure and extended under the rib margin on the left and probably also on the right. The liver was palpable and apparently part of the mass. The right kidney was palpable. The spleen was not palpable. The inguinal glands were enlarged and hard. The extremities showed varicose veins, but were otherwise negative. There were internal and external hemorrhoids.

THE LABORATORY EXAMINATIONS SHOWED:—

The urine was acid, and of 1016 specific gravity, in spite of the small volume. Considerable albumin was present. No casts were found. There was also Acetone and Diacetic Acid present. The 'phthalein output was 15 per cent the first hour and 25 per cent the second hour, a total of 40 per cent.

The blood showed 92 per cent hemoglobin; 3,950,000 red blood cells; 7,000 white blood cells. The differential count was normal. Some poikilocytosis and anisocytosis were noted.

The Wassermann was negative. The blood non-protein nitrogen was 41 mg., uric acid 3.48 mg. and the CO₂ volume per cent 90.61.

Gastric analysis showed no free HCl and a low total acidity. The stool was negative. The electrocardiograms showed a slight tendency to left ventricular preponderance. The gastro-intestinal fluoroscopic X-ray examination showed a hyposthenic individual with a contractural lesion involving the entire stomach. ?Lues, linitis plastica, or scirrhus carcinoma were considered possible diagnoses.

COURSE IN THE HOSPITAL. She vomited small amounts at all times. Duodenal tube feedings were unsuccessful. Abdominal distention was troublesome. Glucose given intravenously and by rectum was of no avail, and the patient gradually lost ground and died one week after admission.

The **CLINICAL DIAGNOSES** were: Gastric carcinoma, involving the oesophagus, pancreas?, and liver?. Carcinomatosis. Dilatation of the oesophagus. Arterio-sclerosis. Anhydremia. Hemorrhoids. Emaciation. Perforation. Gastric Syphilis?, and Plastic Linitis?.

The **PATHOLOGICAL DIAGNOSES** were: Colloid carcinoma of the stomach with metastases to the lymph nodes about the cardiac orifice. Purulent bronchitis. Congestion of spleen, kidneys and intestine. Emaciation. Healed calcified tuberculosis of lungs and peribronchial lymph nodes. Chronic fibrous pleuritis (left), Arteriosclerosis (moderate), and Thrombosis of the hemorrhoidal veins.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS:

KIDNEYS weighed 220 grams. The capsule stripped readily, leaving a smooth surface. On section, the cortex measured 6 mm. and the glomeruli could be distinguished. The medulla appeared dark and cyanotic.

Microscopically, the tubules and, to a less extent, the capsular spaces contained a granular precipitate.

The **AORTA** showed numerous elevated yellowish plaques.

The **HEART** weighed 220 grams. The mitral orifice appeared small, but measured 9 cm. The segments were only slightly thickened. The aortic valve ring measured 7 cm.; the pulmonary 8.5 cm. and the tricuspid 11.5 cm. There was some thickening of the walls of the coronary arteries. The heart muscle was dark brown in color, but was otherwise not abnormal grossly.

Microscopically, the subepicardial adipose tissue showed a marked fatty atrophy and degenerative changes. The myocardial cells showed an advanced simple necrosis, some cloudy swelling and in some places in the myocardium there was fatty atrophy and an infiltration of mononuclear leucocytes. There was also a marked increase of fibroblastic tissue in the myocardium. The blood vessels showed sclerosis and were congested.

CASE 28—(49-77)

A married woman, aged 33, was admitted with the complaint of cough and shortness of breath.

The **FAMILY HISTORY** was negative and her **PAST HISTORY** was irrelevant. She had had scarlet fever four years before admission.

Her **PRESENT ILLNESS** had begun with dyspnoea and oedema during the late months of pregnancy. She had been admitted on the obstetrical service where she had been delivered of a full term still-born infant under twilight sleep. The delivery had been normal, but the patient had been very dyspnoeic and orthopnoeic during and after the delivery. She had also had oedema of the face, feet and lungs after delivery with tachycardia, gallop rhythm, and a weak radial pulse. She had suffered attacks of paroxysms of coughing, marked dyspnoea, and cyanosis, during which the pulse had become weak and thready.

The **PHYSICAL EXAMINATION** on the obstetrical service revealed fine crackles and increased whisper over the upper right lung anteriorly and all over both lungs posteriorly. The heart was apparently enlarged to the left; there was gallop rhythm, an apical systolic murmur, embryocardia and a tachycardia of 120. There was an intermittent fever rising as high as 103° F. The respirations rose to as high as 40. Later, rales were heard only in the upper right lobe posteriorly. The electrocardiogram showed inverted T-waves in all three leads, thus suggesting the diagnosis of myocarditis.

When admitted to Medical Service 9 days postpartum, the patient was well nourished, but pale, orthopnoeic and with no oedema. Her voice was very weak. The heart was regular at a rate of 150; the second sound was accentuated, and there was a question of presystolic murmur over the base of the ensiform and a systolic murmur at the apex. The radial pulses were very weak. The blood pressure was 130/100.

The respirations were short and quick. Over the left upper anteriorly there were bronchovesicular breath sounds with numerous fine crackles toward the outer part. Over the right upper anteriorly there were bronchovesicular breath sounds with increased whisper and bronchophony. The percussion note over the right upper lobe was short and high. Bronchovesicular breath sounds were heard all over the right back with a few fine crackles. The percussion note over the right back was short and high pitched, becoming dull at the midscapular region, and flat two finger breadths below the angle of the scapula. The breath sounds were exaggerated over the left back.

The abdomen showed slight distention and the liver edge was tender, soft and boggy and palpable down to the umbilicus. Below the liver a mass could be palpated which might have been kidney. There was resistance in the splenic region, but no definite mass was made out.

X-ray stereo of the chest showed the whole right side filled with a finely mottled shadow in which round bodies 3-4 cm. in diameter could be distinctly made out. The left lung showed similar shadows. The outer border of these shadows on both sides was very well defined and limited in the lower two thirds of the lung. The liver shadow showed a dense triangular area on the upper border running upward and outward to a sharp point well above the normal level of the diaphragm. The heart shadow extended 4.5 cm. to the right and 10 cm. to the left. The aortic width was 7 cm.

On the day before the patient's death another chest plate showed much less infiltration of

both lung fields. The right supraclavicular region was occupied by a large homogenous opaque mass. Both hilus regions and lower parts of the lung showed many shadows.

THE LABORATORY EXAMINATIONS SHOWED:—

The urine at first contained large amounts of albumin which gradually decreased. The sediment contained coarsely granular and hyaline casts, much pus, and a few red blood cells. Cystoscopic examination was negative. The 'phthalein excretion was 52 per cent. The non-protein nitrogen was 33.3 mg. and 31.3 mg. just before death, with the uric acid content at 4.19 mg. per 100 cc.

Blood cultures were persistently negative. The tuberculosis complement fixation test was negative. The Wassermann was negative. The leucocytes ranged from 11,000 to 19,000. The sputum was scanty, purulent, at first blood-streaked, negative for tubercle bacilli and decidual cells repeatedly. The electrocardiograms showed a simple sinus tachycardia and abnormal T waves.

COURSE IN THE HOSPITAL (Medicl Service): On the fifth day a swelling of the right side of the neck appeared, with redness and heat and tenderness, and a swelling of arm and hand followed. The next day there was a tenderness in the calf of the left leg.

The patient's dyspnea gradually increased. Several attacks were accompanied by collapse. Cheyne-Stokes breathing developed. There was profound weakness, nausea, and vomiting. The temperature was remittent between 99° and 102° and a tachycardia around 130 persisted. The temperature was 104°, the pulse 150, and the respiration 50 on the night before her death, which occurred the 24th day postpartum.

THE CLINICAL DIAGNOSES were: Acute nephritis of pregnancy, Thrombo-Phlebitis of the left subclavian vein and the left popliteal vein, Acute Myocarditis, Pneumonia in the right upper lobe, Resolving Bronchopneumonia of the lower and the right lower and middle lobes of the lung. Deciduoma of Lung?, Chronic passive congestion of the liver, Abscess of the liver, and septicemia (Culture Negative).

THE PATHOLOGICAL DIAGNOSES were: Chronic passive congestion of the liver Acute splenic engorgement, Dilatation of the heart, a slight pericardial effusion with a bilateral pleural effusion, (350 cc. on the right) Multiple infarctions of the lungs with bronchopneumonia, and thrombosis of aorta and both pulmonary arteries.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS:

The **KIDNEYS** together weighed 300 grams and each measured 12x4.5x3 cm. with the cortex measuring 8 mm. The capsules stripped without difficulty, exposing pale surfaces. The cortex appeared swollen and was mottled by yellowish gray foci. The cut surfaces were pale pink in color. The vessels of the pyramids were congested.

Microscopically, the kidneys were greatly congested. Small infiltrated scars were seen here and there destroying glomeruli and tubules, depending upon the location of the scar. The tubular epithelial cells were swollen and granular.

The **AORTA** showed very little atheromatous change.

The **HEART** was greatly distended with blood and appeared considerably enlarged. The muscle weighed 320 grams. There were petechial hemorrhages in the epicardium. The valve leaflets were normal and no dilatation of the orifices was noted.

Microscopically, the subepicardial adipose tissue was very much increased in amount. The myocardial cells showed some cloudy swelling and in some places in the myocardium there was an infiltration of mononuclear leucocytes. There was a beginning proliferation of fibroblastic cells in the myocardium.

CASE 29—(57-67)

A married woman, aged 26, was admitted with the complaint of severe pain in the right chest.

The **FAMILY HISTORY** and **MARITAL HISTORY** were not significant.

The **PAST HISTORY** was negative except for an attack of influenza two years before admission.

The **PRESENT ILLNESS** was dated from an acute attack of severe, deep pain in the right chest with each inspiration, seven years before admission. The pain had lasted for one day and had then disappeared. There had been no trauma, cough, fever or hemoptysis. A similar attack lasting for two days had occurred two years after the first. The pain had remained localized in the right chest but the frequency and the duration of attacks had increased progressively. One year before admission the pain had become so severe that the patient had been totally incapacitated and had frequently required morphine. The pain had been practically constant for a year before admission. She had had night sweats for several months. There had been palpitation of the heart, and shortness of breath on slight exertion for three or four months. For two weeks she had suffered from excruciating pains on pressure over the long bones and the pain in the chest had become localized in the back just over the right scapula. Some fluid had been removed from the right chest just before admission.

The **PHYSICAL EXAMINATION** showed a pale, sallow, emaciated young woman. The skin was yellowish and the mucous membranes were pale.

The veins of the right side of the neck were considerably engorged.

The thorax was deep and somewhat barrel-shaped with quite prominent anterior costal arcs, especially in the upper chest. There was a conspicuous lag over the whole right side. Pus drained from a 12 cm. long incision in the eighth intercostal space in the right axillary line.

The percussion note was flat over the right apex, upper lobe and axilla. The chest was otherwise hyperresonant. The breath sounds were intensely tubular over the flatness and puerile throughout the rest of the lung. The whispered and spoken voice sounds were exaggerated and the tactile fremitus was increased over the region of the flatness.

HEART: The cardiac impulse was in the 5th intercostal space 8.5 cm. to the left. It was forceful and quite localized. A systolic shock and thrill, and a presystolic thrill were felt over the mitral area. The pulmonary second sound was accentuated and the first sound in the mitral area was loud, but not snapping in quality. Auscultation revealed an

apical diastolic rumble and a blowing poorly transmitted systolic murmur in the same area. The blood pressure was 94 systolic and 79 diastolic. The pulse was small, regular and rapid averaging about 96 per minute. The abdomen was flat and tenderness was present along the costal margin. The extremities were negative.

The LABORATORY EXAMINATIONS showed: The urin contained a very slight trace of albumin, a specific gravity of 1010 to 1012 with leucocytes and epithelial cells in the sediment. The 'phtalein output was 60 per cent in two hours.

The blood showed a severe secondary anemia with a hemoglobin of 65 per cent and 60 per cent and a red count of 3,200,000 and a leucocytosis of 9,500 and 12,700. The differential counts varied considerably, at first the polymorphonuclears were 86 per cent, the large mononuclears and transitionals were 4.5 per cent and lymphocytes 9 per cent. The later count showed 70 percent polymorphonuclears, 24 percent large mononuclears and transitionals and 4 per cent lymphocytes. The blood Wassermann was negative.

The electrocardiograms showed right ventricular preponderance.

An X-ray plate showed the heart pushed over the left with the right border, 1.5 cm. to the left of the midline in the 5th intercostal space. The left border was 5 cm., 8cm., 12 cm. and 14 cm. to the left in the II, III, IV and V intercostal spaces.

COURSE IN THE HOSPITAL: She was given 1250 cc. of whole blood by the Lindeman method. Four rubber needles were buried in the lung tumor for 20 hours at each of two treatments. The T. P. R. chart showed a T. of 100.5°, P. 110 to 130 and R. 20 to 32.

The patient gradually got around and died eight weeks after admission.

The CLINICAL DIAGNOSES were: Massive carcinoma of the left lung. Severe secondary anemia. Mitral stenosis.

The PATHOLOGICAL DIAGNOSES were: Carcinoma of the right lung with infiltration of the right and left pleura, chest wall, diaphragm, surface of the liver and adrenals. Abscess of the right pleural cavity. Bronchopneumonia of the left lung. Fatty degeneration of the liver and kidneys. Petechial hemorrhages of the skin and bladder mucosa. Slight chronic mitral endocarditis. Cyst of the broad ligament. Accessory spleen.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS:

KIDNEYS: Together weighed 220 grams and were of equal size. The capsules stripped easily, showing smooth normal-appearing kidneys. On section, the cortex measured 6 mm. and the glomeruli were well seen.

Microscopically, the spaces between Bowmans capsule contained cellular debris. In many instances the capsule was broken. The capillaries of the glomeruli contained a few red blood cells. The cells of the glomeruli were granular and pink staining. The cell outlines of such endothelial cells were lost. The tubules contained granular and pink staining material. The lining cells were ragged, granular and pink staining. Some tubules showed a few necrotic cells and fibrous stroma.

The AORTA was moderately elastic.

The HEART: The pericardium appeared normal and contained only a small amount of fluid. The heart was small, weighing only 135 grams, but it was otherwise apparently normal externally. There was slight thickening of the lateral flap of the mitral valve. The hydrostatic tests were negative. The valve rings measured as follows: The mitral 6.5 cm., the tricuspid 8.5 cm., the pulmonary 6.5, the aortic 6.5 cm.

Microscopically, there were small granular blue staining masses which appeared to be made up of bacteria. These were confined to the blood vessels, however. Some of the myocardial cells showed an advanced cloudy swelling which in some cases went over to simple necrosis, especially along the subepicardial adipose border. There was a diffuse round cell infiltration in areas immediately beneath the endocardium.

CASE 30—(35-91)

An unmarried female, aged 33, was admitted with the complaints of chills and fever for five months, a loss of appetite, weight, strength, and color.

In her PAST HISTORY she had had scarlet fever and, during convalescence, the physician noticed a loud heart murmur. Her heart had never been examined before, so that she did not know whether the lesion causing the murmur had been present previous to the scarlet fever or not. At any rate she had never previous to, at the time of, or after the infectious disease, had any cardiac symptoms.

The PRESENT ILLNESS had begun five months before admission with a sudden onset of fever and chills, the latter had occurred about every third day at different times of the day. Within a month the chills had begun to occur every day. At about the same time she had begun to have severe pains in the left side, at the base of the axilla and in the left upper quadrant, or the splenic region. She had been in a hospital for six weeks, during which time a diagnosis of pleurisy had been made. The symptoms had subsided, but she had not regained her strength. She had journeyed North for the summer and while there her chills had begun again. Blood cultures had failed to yield any organism. The chills and fever had persisted, the patient had lost her appetite, had lost weight and strength and had become pale and sallow. The chills had become less severe, but the fever had increased to 104°-105° and nausea and vomiting had become troublesome.

The PHYSICAL EXAMINATION showed pallor with slight sallowness and some emaciation. The mucous membranes were pale and slightly cyanotic, but no petechiae were present. Two carious teeth and many apical abscesses were discovered. The tonsils were enlarged and apparently septic. There was an oozing epistaxis. The neck was negative. The lungs were resonant and clear.

The HEART was apparently not enlarged. The dullness extended 3.5 cm. to the left and 10 cm. to the right of the midsternal line. The aortic dullness measured 5 cm. in width. There was a systolic thrill over the manubrium, most intense in the mid-sternal line. The apex impulse was in the 5th intercostal space, 10 cm. to the left of the midsternal line. A loud rough systolic murmur could be heard throughout the chest, but was loudest over the base, with the point of maximum intensity in the midsternal line, at the level of the 3rd intercostal space. The heart sounds were normal. Occasional extrasystoles were noted,

but the mechanism was otherwise regular. The blood pressure was 120-148/70-75. The pulse was normal.

Abdominal palpation revealed nothing but tenderness in both upper quadrants and in the flanks on deep pressure. Neither the spleen edge nor the liver could be definitely felt, although there was considerable resistance in each region.

Extremities: The finger tips were slightly but definitely clubbed and faintly cyanotic. No petechiae were found.

The LABORATORY FINDINGS were quite significant.

Blood cultures were repeatedly negative. The blood showed 70 per cent hemoglobin, 3,800,000 red cells, and 25,000 white cells. The differential count showed 90 per cent neutrophilic polymorphonuclear cells. The Wasserman was negative. The blood non-protein nitrogen was 91 mg. per 100 cc.

The urine contained a large amount of albumin, blood and pus. No casts were found. The phthalein output was zero in two hours. Ureteral catheterizations yielded pus containing Gram-negative bacilli from each kidney.

A heart X-ray plate showed a shadow normal in shape and size. Films of the teeth revealed five apical granulomata.

The electrocardiogram recorded a few extrasystoles, but no definite ventricular preponderance.

COURSE IN THE HOSPITAL: Urotropin was given by mouth and the kidneys were treated locally through the ureteral catheter and an autogenous vaccine from the urine culture was administered. The chills subsided but the fever continued between 99.5° and 103°. The patient was difficult to feed because of an obstinate nausea. Attacks of epistaxis continued to come on. The anemia increased in severity until the examination showed only 30 per cent hemoglobin, 1,800,000 red cells, and 9,000 white cells. The blood non-protein nitrogen gradually rose to 208 mg. per 100 cc. and uremic stupor intervened. A blood transfusion afforded temporary relief. A gangrenous stomatitis developed. The patient continued to grow weaker and more comatose and died six weeks after admission.

The CLINICAL DIAGNOSES were: Pylonephritis, congenital heart disease, severe secondary anemia, gangrenous stomatitis and uremia.

The PATHOLOGICAL DIAGNOSES were: Suppurative nephritis with multiple abscesses, chronic glomerular nephritis with a large smooth kidney, congenital heart disease with a patent interventricular septum and foramen ovale, fibrinous pericarditis with pericardial exudation, subepicardial hemorrhages, petechial hemorrhages into the stomach, hypertrophy of the spleen with focal necrosis, fibrinous perisplenitis and perihepatitis, secondary anemia, gangrenous stomatitis.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS:

KIDNEYS: The right kidney weighed 200 grams and measured 12.5x6x3.75 cm, the left weighed 220 grams and measured 12.5x6x4 cm. The cortex of each averaged 9 mm. The capsules stripped with ease leaving opaque, pinkish-gray surfaces over which were seen numerous opaque, yellow slightly elevated areas of 3 to 4 mm. in diameter. On section the substance of the kidney resembled the surface in color and the opaque yellow areas were filled with thick-caseous-like material. The cortical striations and the medullary rays were easily made out; both were rather pale, but the line of demarcation between the cortex and the medulla was fairly distinct. The kidney pelvis had a slightly granular-like deposit on its surface.

Microscopically, the kidneys showed a marked sclerosis of the glomerulae; in some instances the glomerulae were completely sclerosed, the capsular space obliterated and the entire structure changed to a hyaline mass of connective tissue. In some instances there was a proliferation of the epithelium lining Bowman's capsule. This mass of cells of crescentic shape entirely filled the capsular space. The tubules throughout the space, were dilated, in many instances this dilatation was marked. The epithelial cells lining the tubules were flattened and the lumen contained granular pink-staining debris. An occasional hyaline cast was seen. There were areas of round-cell infiltration. These cells were collected in small groups. The blood vessels showed no sclerosis.

The AORTA was quite elastic and free from any gross change. There was a small nodular bulging of the intima at one point; this consisted of a thickening of the intima with little evidence of degenerative change.

The HEART: The pericardial fluid was increased in amount and cloudy from flakes of fibrin. The epicardium showed areas of fibrinous deposit and evidence of inflammation. The valves were smooth and thin and normal in every way. The valve rings measured as follows: Aortic 7 cm., mitral 10 cm., pulmonary 6 cm., and tricuspid 11 cm.

The foramen ovale was patent, but there was a flap-like structure on the right. This fit very closely to the interauricular septum and the opening on the left side was 2 cm. further along the septum.

There was an opening through the membranous portion of the interventricular septum. This opening measured about 6 mm. in diameter and allowed fluids to pass quite readily from one ventricle to the other. The thebesian valve was somewhat enlarged.

Microscopically, throughout the myocardium there was a marked infiltration of mononuclear leucocytes, a large number of which were of the plasma cell type. There was some fibroblastic proliferation, which indicated a chronic myocarditis. The subepicardial connective tissue and fat was likewise affected and the epicardium was much thicker than normal. The muscle cells on the whole were slightly smaller than normal and in some parts showed cloudy swelling.

CASE 31—(11-11)

A man, aged 63, was sent into the hospital with the X-ray diagnosis of carcinoma of the oesophagus and colonic obstruction above the pelvi-rectal juncture. He was transferred to surgery for the purpose of doing an exploratory laparotomy and probably a colostomy for intestinal obstruction.

His **CHIEF COMPLAINT** was pain in the abdomen with constipation, vomiting and difficulty in swallowing.

The **FAMILY HISTORY** was negative.

In his **PAST HISTORY** he had had typhoid fever at six, and complete recovery. He had had gonorrhea, with a bubo in the right inguinal region. Syphilitic infection was denied. He had used 12 to 15 glasses of beer per day and considerable tobacco each day for many years.

His **PRESENT ILLNESS** probably had its onset three years before admission, when he had had a severe attack of pains and cramps in the lower abdomen and obstinate constipation, which had been diagnosed "Inflammation of the Bowels." Relief came immediately after movement of the bowels, but he remained in bed for two weeks.

A dull pain in the lower abdomen had returned two months before admission. The pain was again accompanied by obstinate constipation. The pain had increased in severity from day to day and had spread all over the abdomen, but had been more severe in the lower quadrants. There had been no vomiting. The bowels had not moved for four days after the onset of the pain. The stools had been small, dry and nodular. The pain had been relieved after movements of the bowels.

At the same time that he had noted the abdominal symptoms he had also noticed difficulty in swallowing with immediate regurgitation of foods. Even liquids had not been tolerated while the patient had abdominal symptoms, but otherwise liquids had passed; no solids had escaped regurgitation, however.

There had been a second onset of abdominal symptoms two weeks before admission, with gurgling and rumbling and absolute constipation, which had persisted. The pain too had been constantly present. The dysphagia had not increased.

The **PHYSICAL EXAMINATION** showed: Emaciation, cachexia, pyorrhea and pharyngitis. The neck showed slight enlargement of the posterior cervical glands. Thorax: The percussion note was hyperresonant except for slight dullness in the right back on a level with the 8th rib, and rising slightly to the postaxillary line. There was besides slight dullness decreased breath sounds and voice sounds. The breath sounds were harsh elsewhere with a prolonged expiratory murmur.

The veins laterally were distended and extended over the abdomen.

HEART: The cardiac impulse was in the 4th intercostal space in the nipple line. The aortic second sound was louder than the pulmonary second sound. The blood pressure was 140/75. The radials and brachials were palpable and tortuous. The pulse was weak.

The Abdomen was distended and bulging in the flanks. There was visible and palpable peristalsis, gurgling and succussion all over the abdomen. There was dullness in the right flank and tympany in the left abdomen extending 8 cm. above the pubis. The liver and spleen were not felt and the dullnesses were obliterated. There was a left inguinal hernia.

Genitals: The right testicle was much larger than the left, very hard with a nodular, irregular surface.

Rectal examination was painful, mucous was present and pus on the examining finger. There was felt the hard right lobe of the prostate.

The extremities showed varicose veins, sclerotic vessels and active reflexes.

At operation about 100 c.c. of blood stained ascitic fluid was noted. A mass the size of a small egg was found at beginning of the rectum on the level with the pelvic brim. The mass caused puckering of the rectum. A number of large glands were found along the aorta. The colon was friable. A left inguinal colostomy was done.

The **LABORATORY EXAMINATIONS:** The urine at times contained a trace of albumin and showed a few hyalin casts. The blood showed a secondary anemia with a leucocytosis of 11,200 to 19,720.

The electrocardiograms showed low T waves, suggesting myocarditis.

COURSE IN THE HOSPITAL: The patient lost ground progressively. He developed a purulent bronchitis and cyanosis. The pulse was irregular at times and thready.

The **CLINICAL DIAGNOSES** were: Carcinoma of the oesophagus and rectum. General arteriosclerosis. Chronic nephritis. Simple anemia. Chronic intestinal obstruction. Femoral hernia. Cellulitis of the abdominal wall. Acute bronchitis. Chronic fibrous pleurisy. Spontaneous rupture of the oesophagus.

The **PATHOLOGICAL DIAGNOSES** were: Squamous cell carcinoma of the oesophagus at the bifurcation of the trachea, with metastases to the regional lymph nodes. Perforation with peri-oesophageal abscess. Adenocarcinoma of the rectum with metastases to the pelvic lymph glands. Colostomy. Subcutaneous abscess of the abdominal wall. Chronic localized fibrous pericarditis. Bilateral bronchopneumonia. Acute splenic enlargement. Arteriosclerosis. Dilatation of the ascending aorta. Chronic passive congestion of the viscera. Multiple diverticuli of the small bowel.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS:

KIDNEYS: They were both very small. The right weighed 105 grams, while the left weighed 110 grams. The capsules stripped easily. The organs were soft and in section appeared to have the same dark red color.

Microscopically, some of the tubules showed hyalinization. There were a few small areas of scar tissue. The vessel wall were thickened.

AORTA: The ascending portion of the arch was definitely dilated. There were numerous irregularly raised areas to be seen on the inner surface of the upper portion of the aorta. There were numerous fibrous and fatty plaques in the intima. The walls of the coronary arteries were thickened and the lumina stood open.

HEART: The entire heart had a homogenous yellowish pink color due to extensive adhesions between the pericardium and the surface of the heart. The valve rings measured: Mitral 9 cm., aortic 8.5 cm. tricuspid 11 cm., and pulmonary 8 cm. The mitral valve was uniformly slightly thickened. There was a small yellow plaque near the edge of the aortic valve. The valves were otherwise normal. The endocardium appeared normal. The myocardium grossly showed nothing of interest.

Microscopically, the myocardium was very compact and in places the cell outlines could scarcely be made out. There were areas of marked cloudy swelling and simple necrosis

throughout the sections. The endocardium was, in places, thicker than normal. There was much fibroblastic proliferation throughout the sections. The blood vessels were congested and the walls were thicker than normal. Some of the muscle cells showed cones of golden yellow hemofuscin pigment extending from the poles of the nuclei, the evidence of brown atrophy of the heart muscle.

CASE 32—(30-96)

A man, aged 34, was brought to the hospital because of unconsciousness. The FAMILY HISTORY was not important. The PAST HISTORY was of some importance. He had had diphtheria twice, scarlet fever once and frequent sore throats but no rheumatic fever or chorea. There was no history suggesting syphilis. The patient had had a chronic discharging ear since the age of 14.

The PRESENT ILLNESS had begun two weeks before admission, with pain in the region of the right ear. The pain increased progressively in severity. Early on the morning of admission he had vomited, had become delirious and had lost consciousness.

The PHYSICAL EXAMINATION showed unconsciousness. The mastoid region was tender on the right side. The right ear discharged pus. The neck was stiff and there was moderate opisthotonus. The knee kicks and arm reflexes were exaggerated. There was a positive Babinski on the right and left sides. There was no cyanosis, dyspnoea or engorged neck veins. The arteries were thickened. The heart was not enlarged. No murmurs were heard. There were no thrills or shocks. The blood pressure was 135/65.

The LABORATORY EXAMINATIONS showed: The urine contained much albumin, but no casts. The leucocytes were increased to 31,000; the blood was otherwise normal. The electrocardiogram was normal.

He was operated for acute mastoiditis and developed pneumonia seven days later and died.

The CLINICAL DIAGNOSES were: Acute right mastoiditis, Abscess of the brain, Cerebrospinal meningitis, Bronchopneumonia, Acute bronchitis, Serofibrinous pleurisy. Chronic right otitis media.

The PATHOLOGICAL DIAGNOSES were: Purulent right otitis media and mastoiditis. Brain abscess in the right temporal lobe. Purulent cerebrospinal meningitis. Oedema of the brain. Fibrous pleural adhesions. Left sided empyema. Massive bilateral bronchopneumonia. Purulent bronchitis. Cloudy swelling of the liver and kidneys. Acute splenic tumor. Calcified tuberculous nodules of the left lower lobe and peribronchial lymph nodes. There was a complete double ureter on the right. Cultures showed *Staphylococcus aureus*. *Pneumococcus* type II and *B. Pyocyaneus*.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS:

KIDNEYS: The right kidney weighed 140 grams and measured 13x7x3 cm. The left was about the same in size. The stielate veins were injected and the glomeruli prominent. There were some gray opaque streaks along the outer tubules. There was a complete right double ureter with a double pelvis and a double opening of the ureters into the bladder; one above the other.

Microscopically, there was a marked cloudy swelling. The tubular epithelium was swollen and granular.

The AORTA at its root had a few yellowish white patches. Microscopically, no change was noted.

The HEART weighed 320 grams and externally appeared normal. The valves measured as follows: Aortic 7.5 cm., mitral 10.5 cm., pulmonary 7 cm., tricuspid 11.5 cm. The corpus arantii of one aortic cusp was thickened. There were fine fenestrations at the edges of the aortic and pulmonary valves.

Microscopically, the subepicardial fat was increased in amount. The myocardium along this showed a rather marked fatty atrophy. In some places the subepicardial fat and myocardium contained groups of polymorphonuclear and mononuclear leucocytes. There was a slight increase in connective tissue especially around the blood vessels. The endocardium showed no marked changes. The presence of fibroblastic proliferation indicated a chronic process.

CASE 33—(46-80)

An unmarried woman, aged 54, was admitted, with a complaint of a growth in the left breast.

The FAMILY HISTORY and PAST HISTORY were unimportant. The patient had passed the menopause ten years previous to admission. She gave no history of rheumatic fever, chorea, scarlet fever, diphtheria or syphilis. She had had scarlatina and pertussis in childhood without complications.

The onset of her PRESENT ILLNESS was not definitely known. The growth had been present in the breast for a number of years. It had caused very little pain or trouble until it had become ulcerated two months before admission.

The PHYSICAL EXAMINATION showed nothing abnormal except the ulcerating tumor of the left breast.

The arteries were not thickened. There was no dyspnoea, cyanosis or engorged neck veins. The blood pressure was 110/75. The left border of the heart could not be made out owing to the ulcerating tumor of the left breast. There was no enlargement to the right. No murmurs and no thrills were noted.

LABORATORY DATA.

The urine was always negative. The blood findings were within normal limits. An electrocardiogram showed sinus tachycardia.

COURSE IN THE HOSPITAL: The patient underwent a radical breast amputation and went into shock a few hours afterward. She was transfused. She developed toxic symptoms shown first by tachycardia and stupor. The collapse was not due to cardiac failure.

No digitalis was given. She died on the fifth day after operation, that is twenty days after admission.

The CLINICAL DIAGNOSES were: Carcinoma of the left breast, axillary and supraclavicular lymph nodes and the liver. Shock. Burn of arm and right shoulder. Acute osteomyelitis of a rib. Bronchopneumonia. Abscess of the lung. Chronic fibrinous pleurisy. Inflammation of the left parotid gland.

The PATHOLOGICAL DIAGNOSES were: Carcinoma of mammary gland, axillary and supraclavicular peribronchial and gastrohepatic glands. Carcinomatous metastasis to the liver. Osteomyelitis of the ribs in the operative field. Bronchopneumonia. Abscess of the lung. Old peritoneal adhesion. Thickening of the pleura with hyaline degeneration. Healed pulmonary tuberculosis. Calcified tubercles of the spleen. Blood cultures showed a hemolytic streptococcus.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS.

KIDNEY: The right kidney weighed 160 grams and measured 12x6x2.5 cm., the left weighed 150 grams and measured 10.5x6x3 cm. The cortex measured 6 mm. in each. The capsules stripped with moderate ease leaving a finely granular surface which was torn at one of two points. The stellate veins were not at all prominent. On section, the kidneys appeared swollen. The cut edges tended to turn back and the cut surfaces were quite pale and had a greasy glass appearance.

Microscopically, the cells lining the tubules were very much swollen and quite granular. Sudan III showed fat. There was a moderate congestion of all capillaries.

The AORTA was quite elastic and appeared normal microscopically.

The HEART was about normal in size, weighing 205 grams. The valves all had delicate edges except the aortic, one cusp of which had a thickened corpus arantii, which stood 2 mm. above the surface. The valves measured as follow: Aortic 8 cm., mitral 9.5 cm., pulmonary 7.5 cm., and tricuspid 10.5 cm.. The heart muscle appeared normal.

Microscopically, throughout the myocardium were many small and large irregular finely granular blue staining masses around which there were very dense infiltrations of polymorphonuclear leucocytes. In some places liquifaction had taken place and abscesses had formed. These foci were present throughout the sections, some in the epicardium, some in the subepicardial fat and some in the myocardium. The endocardium presented a thin growth of this finely granular material indicating an endocarditis. There was cloudy swelling in the zones around the abscesses and a fibroblastic proliferation evidencing chronicity. Colonies of bacteria were seen in the blood vessel suggesting hematogenous metastases of the organism.

CASE 34—(34-92)

An Italian, aged 38, was admitted with the complaint of distended abdomen and pain in the lower left quadrant.

The FAMILY HISTORY and PAST HISTORY were unimportant, as far as could be determined. He recalled no childhood disease.

The PRESENT ILLNESS was considered by the patient to have had its onset only 4 weeks previous when his abdomen had begun to swell and caused shortness of breath. He also noted a loss of weight and appetite, vomiting and a deepening of the brownish pigment in the skin. There had been pain in the left lower quadrant before the ascites and had become pronounced.

The PHYSICAL EXAMINATION showed a cachectic individual, propped up in bed, dyspnoeic and slightly orthopnoeic. Thorax: The lungs were negative. The rib margins flared out. The heart apex impulse was seen and felt in the 5th intercostal space, 9 cm. from the midsternal line. No thrills were felt. The character of the sounds was good. The arteries were sclerosed. The blood pressure was 118/94. There was a marked ascites. A mass was felt per rectum and one also in the epigastrium.

The LABORATORY FINDINGS: The urine was negative. The phthalein output was 60 per cent in two hours. The gastric fractional analysis showed an achlorhydria. The blood showed: Hemoglobin 40 per cent, red blood cells 2,760,000, white blood cells 11,000. The blood Wassermann was negative. The electrocardiogram showed no muscle preponderance and the Q. R. S. deflections were of low amplitude. An exploratory operation was done the patient died five hours later, that is, two weeks after admission.

The CLINICAL DIAGNOSES were: Carcinoma of the stomach and peritoneum. Severe simple anemia. Ascites. Multiple chronic lymphadenitis. Carcinoma of the omentum.

The PATHOLOGICAL DIAGNOSES were: Colloidal carcinoma of the stomach with metastasis to the omentum and peritoneum. Bloody ascites. Cloudy swelling of the kidneys. Old pleural adhesions. Calcified tuberculous nodules in the right lung. Healed tubercles in the spleen.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS.

KIDNEYS: The right kidney weighed 130 grams and measured 10x6x3.5 cm. and the cortex measured 8 mm. The left kidney was of similar size. The cut surfaces appeared slightly cloudy and the edge tended to roll back. The capsules stripped with moderate difficulty tearing the surface in many places. The surfaces were of a pinkish green color and on section the markings of the cortex were made out with ease.

Microscopically, the kidneys showed wide tissue spaces denoting considerable oedema. The tubules and also the inside of the capsule of some glomeruli showed considerable pink staining material.

The AORTA appeared normal to gross and microscopical examination.

The HEART muscle was rather dark and weighed 225 grams. The valve rings measured as follows: Aortic 9 cm., mitral 10 cm., pulmonary 8 cm. and tricuspid 12 cm. The tricuspid showed a number of thickenings at the insertion of the chordae tendinae.

Microscopically, throughout the sections the myocardial cells showed an advanced cloudy swelling. The subepicardial fat showed some signs of inflammation. There was a definite increase of fibroblastic tissue.

CASE 35—(21-103)

An Italian boy, aged 16, was brought into the hospital because of delirium and fever. The FAMILY HISTORY and PAST HISTORY were irrelevant.

The PRESENT ILLNESS had begun with a headache two days before admission, but the patient had continued to work in spite of feeling ill. He had returned home feeling quite ill and had gone to bed where he had vomited frequently. He had only remained in bed through the night, for he had felt very good and had been bright and happy on the morning of the day of admission. At 11 A. M. he had had a convulsion, after which he moaned and muttered. He had not been able to speak or recognize anyone or anything. He had rapidly become worse, the delirium had increased and he had been brought to the hospital.

The PHYSICAL EXAMINATION showed a fairly well nourished boy twisting and tossing restlessly in bed, uncomfortable in any position, moaning and occasionally assuming an opisthotonus position. The neck was rigid. The Kernig and Brudzinski signs were positive. The pupils were dilated and fixed to light and the accommodation test was not accomplished.

CHEST: The breathing was stertorous and irregular and Biot's in type. The lungs were negative.

HEART: The heart sounds were clear, regular and slow at times. At other times the heart was overactive at a rate of 100 per minute. It was not enlarged. The mitral first sound was loud and snapping.

ABDOMEN: The liver and spleen were not palpable; no masses were felt. There was no rigidity or tenderness.

EXTREMITIES: There was a soft fluctuating swelling of long duration over the right hip. The patient had limped for some time. The K.Ks and A.Ks were not obtained. They were absent or very sluggish.

The LABORATORY EXAMINATIONS showed the urine to be turbid and concentrated and to contain no sugar, albumin or sediment. The blood showed 85 per cent Hb., 5,150,000 r. b. c. and 5,440 w. b. c. The Wasserman was negative. The electrocardiogram showed a right ventricular preponderance. The spinal fluid showed many meningococci and leucocytes.

COURSE IN HOSPITAL: The patient was given antimeningococcus serum in amounts of 30 c.c. every 8 hours intravenously. The patient became rational the day after admission, but the temperature remained high. He became drowsy.

The left optic disc was swollen 2 diopters and there was hemorrhage in the inner lower quadrant. Intraventricular puncture was done and serum administration carried on through the puncture. There developed tracheal rales, cyanosis, and dullness at the bases with rales and tubular breath sounds. Bronchopneumonia and oedema of lungs were present. The heart sounds were clear and the action was normal.

The CLINICAL DIAGNOSES were: Cerebrospinal meningitis; Bronchopneumonia.

The PATHOLOGICAL DIAGNOSES were: Cerebrospinal meningitis, bilateral bronchopneumonia, chronic fibrous pericarditis, chronic passive congestion of the liver and kidneys, chronic perisplenitis and perihepatitis, focal caseous tuberculosis of the spleen and the right hilus glands, chronic fibrous pleurisy of the right, cloudy swelling of the kidneys, fatty liver, and lymphoid hyperplasia of the spleen.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS.

KIDNEYS. The left kidney weighed 120 grams and measured 11x5x2 cm. The right weighed 120 grams and measured 11x6x2 cm. The two kidneys had the same appearance. The capsule stripped easily, exposing a pale surface on which numerous engorged capillaries could be seen. The cortex was fairly regular and measured 7 mm. The normal kidney markings could be distinctly made out. The tubules were unusually yellow in color.

Microscopically, the tubular epithelium was swollen and granular. The nuclei of the cells were often unstained. The vessels were engorged.

The HEART showed no gross abnormality except a soldier spot, an area of chronic fibrous pericarditis. The valves were all normal in size and texture.

Microscopically, the entire myocardium showed a diffuse mononuclear leucocytic infiltration with foci here and there. Masses of bacteria were present in the vessels. The endocardium was very much thickened. The epicardium in certain areas showed fibrous thickening with some hyalinization of the connective tissue.

CASE 36—(25-170)

A negro preacher, aged 46, was admitted with the complaint of severe shortness of breath and failing health for a year.

In his PAST HISTORY he had had gonorrhea at 14. Syphilitic infection was denied. He had influenza in 1918, and frequent "cold on lungs" each winter. He had been married 11 years and his wife had never become pregnant, and had died in 1919 of "kidney trouble."

His PRESENT ILLNESS had begun eight months previously with a tight dry cough and a "cold" in the upper part of chest. A cough had continued from the onset until about a month before admission. Three months before admission he had had an acute attack of dyspnoea while eating. The attack had passed off, but a rattling and feeling of closing up of the throat had remained. Similar attacks had recurred, becoming so severe that the patient became orthopnoeic. Hoarseness had been present for three months. An acute attack had begun six days before admission. This had been relieved some by smoking "asthma powders."

PHYSICAL EXAMINATION showed orthopnoea and shallow, wheezing expiration. The patient had a husky voice, a hoarse cough and speech difficulty. There was tracheal tug. Systolic and diastolic shocks were felt over the right side of the manubrium. A systolic heave was felt over the sternal end of the right clavicle, manubrium and upper chest. The neck veins were engorged. The retromanubrial dullness measured 10.5 cm. The left border was 11 cm., and the right border 5.5 cm. from the midsternal line. The

heart sounds were distinct, no murmurs were heard. Squeaking, whistling expiration was heard over the back. The pulse seemed weaker on the right. The blood pressure was 135/80 on the left and 125/80 on the right.

The LABORATORY TESTS showed: The urine was negative. The blood, red blood cells 3,800,000; white blood cells 6,800; Hg. 70 per cent. The non-protein nitrogen was 30.5 mgm. per 100 cc.. The Wassermann was negative. The electrocardiogram was normal. Chest X-ray plate showed the shadows of the great vessels broadened.

COURSE IN THE HOSPITAL: The dyspnoea increased and the attacks became very severe. Morphine was frequently necessary.

9 P. M. on the day before death a very severe attack began which was not relieved by morphine. At 1 P. M. of his last day he was relieved by oxygen, only to stop breathing 15 minutes later. Tracheotomy was done without anaesthesia. Artificial respiration was given. A pulsating mass was felt obstructing the trachea completely from the right side. A stiff rubber tube was inserted and the patient was revived. The patient died about 10 hours later, exhibiting a few gasping respirations and a gradually weakening pulse. He had been in the hospital exactly two weeks.

The CLINICAL DIAGNOSES were: Aneurysm of the transverse arch and the innominate artery. Syphilis Traumatic cataract of the left eye. Caries of the teeth and pyorrhea.

The PATHOLOGICAL DIAGNOSES were: Aneurysm of the innominate artery. Syphilitic aortitis. Thrombus in the aneurysm plugging the channels to the carotids and right subclavian. The vagi and recurrent laryngeal nerves were imbedded in the aneurysmal wall. Aneurysm of the descending aorta with erosion of the 6th thoracic vertebra. Pseudomembranous tracheitis. Caseous and fibrous peribronchial lymph nodes. Anthracosis of the lungs. Healed tubercles of the liver. Atrophic cirrhosis of the liver. Prostatic hypertrophy.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS.

KIDNEYS: The left kidney weighed 210 grams and measured 13x7x6 cm. with a cortex of 10 mm. The right weighed 150 grams and measured 11.5x6.5x4 cm. with a cortex of 7.5 mm. The capsules stripped with moderate ease tearing the kidney substance in a few places. On section, the color was pinkish gray. The cortical striations were not distinct, the surface had a slightly cooked appearance.

Microscopically, the outlines of the cells lining the tubules were broken and in many instances only the basement membrane of the cells remained. The lumen contained casts of a homogeneous nature, taking the pink stain. Bowman's capsules were intact, but within the capsules of a few glomeruli similar in nature to those seen in the tubules. The blood vessels at their entrance into the glomerular tufts showed rather marked hyalin changes.

The AORTA was very inelastic. The intima was very rough with numerous puckering and longitudinal folds. Microscopically, the intima was markedly thickened. The fibers of the media in many places were broken and replaced by scar tissue with considerable round cell infiltration. The adventitia was rather extensively infiltrated. The adventitia was rather extensively infiltrated with round cells especially about the blood vessels. No typical atheromata were found and only one small calcium deposit was noted.

The innominate artery presented a pear-shaped aneurysm measuring 11x9x6 cm., the anterior posterior diameter was the smallest. The right subclavian and carotid arose from the upper extremity of the aneurysm. The aneurysmal wall microscopically was made up of scar tissue and typical specific atheromatous lesions. The vessel wall was totally degenerated and lined with a laminated thrombus.

The orifices of the coronary vessels were patent but irregular and hard.

Heart: The ventricles alone weighed 200 grams. The valves appeared to be capable of functioning normally. The aortic valve appeared slightly thick and inelastic. The corpora arantii were thickened and stood out from the thickened and irregular surfaces of the valve about 2 mm. The mitral valve was somewhat thickened and nodular along its free edge and on one leaf there were two small hard yellow nodules 1 mm. in diameter. The tricuspid valve was slightly thickened at its free border. The pulmonary valve appeared normal. The valve measurements were, mitral 10 cm., tricuspid 12 cm., pulmonary 9 cm., and aortic 10 cm.

Microscopically, there was only a slight increase in connective tissue. The blood vessels were congested and showed very little or no conspicuous perivascular change. There was some mononuclear and polymorphonuclear leucocytic infiltration in the myocardium. The cells of the myocardium showed an advanced cloudy swelling.

CASE 37—(32-94)

A man, aged 31, was admitted with the chief complaints of weakness and loss of color. In his past history he had had gonorrhea at 17, with a sore on the penis at the same time. Typhoid fever at 25, had been complicated by an acute nephritis. With this he had been in bed 7 months.

The PRESENT ILLNESS had begun insidiously about 10 weeks before his first admission. He first had noticed sleepiness and malaise, in that he had been tired all the time no matter how much sleep he had had. The symptoms had gradually increased. He also had had headaches, palpitation and dyspnoea. He had been despondent and paranoiac.

PHYSICAL EXAMINATION showed a lemon-yellow tint to the skin, and pallor. The general panniculus was thick. The patient was apathetic. He had caries of the teeth and pyorrhea. The tongue was atrophic. Chest: the lungs were very resonant. The heart was not enlarged, systolic murmurs were heard over the pulmonic and mitral areas. The abdomen was negative. The liver and spleen were not palpable. The neurological examination showed sluggish K. K.'s and A. K.'s and a positive Romberg.

The LABORATORY examinations showed: Blood: R. B. C. 1,232,000; W. B. C. 8,800; Hgb. 43 per cent; normoblasts, megaloblasts, anisocytosis, and poikilocytosis. The blood

improved and on discharge he had: R. B. C. 4,128,000; W. B. C. 6,660; Hgb. 90 per cent. The Wassermann was negative. The stools were negative. The urine was negative except for increased urobilin content. The 'phthalein output was 60 per cent in two hours. He received Fowler's Solution, Bland's pills and a transfusion of 500 c.c. of blood. He was discharged seven weeks after admission.

He was re-admitted one week after discharge with symptoms of a manic depressive psychosis. The neurological findings were: Absent K. K.'s and A.J.'s and a? questionable Babinski and hyperaesthesia. The mental symptoms rapidly cleared up.

LABORATORY: On admission the R. B. C. were 5,432,000; W. B. C. 10,600 Hgb: (?) 95 per cent. There was a definite fall of the R. B. C. count to $3\frac{1}{2}$ million before discharge, 6 weeks after the second admission.

The third admission was 7 months after the second discharge. He had been able to do very little work. His weakness had gradually increased. Physical examination was about as above. The fat was well preserved; the face was slightly puffy. There was slight general pigmentation. The liver was 3 cm. below the costal margin. The spleen was enlarged and firm.

LABORATORY: The laboratory examination showed R. B. C. 550,000; W. B. C. 4,400; Hgb. 25 per cent. Smears showed signs of bone marrow activity. A fractional gastric analysis showed achlorhydria. The duodenal content was pleochromic with bilirubin.

COURSE IN THE HOSPITAL: A transfusion of 500 c.c. of blood was given. There was occasional vomiting, diarrhea and nose bleed. There was epigastric pain, headache and fever of 99 to 101 degrees, with tachycardia of 100 to 120. Two transfusions of 1,000 c.c. each were administered, with a chill and fever after each. After the last one the pulse rose to 140 and the patient was cyanotic and was given atropine and adrenalin. Vomiting and diarrhea together with other complication as acute pleurisy, furunculosis, and asychosis, developed. A remission with an improvement in the blood picture, appeared but soon disappeared, not even a reticulated cell being left.

The neck veins showed slight engorgement and the liver presented a questionable expansile pulsation. A murmur developed over the lower end of the sternum. The concentration-diuresis test showed evidence of nephritis. The 12-hour urine showed a fixed specific gravity and a large night volume, with albumin and casts. The 'phthalein output was 65 per cent in two hours. The patient refused to eat, rapidly grew worse, and died one week after the transfusion that is two months after admission.

The **CLINICAL DIAGNOSES** were: Pernicious anemia. Chronic myocarditis. Acute cardiac dilatation. Relative mitral and tricuspid insufficiency. Chronic interstitial nephritis. Ataxic paraplegia.

The **PATHOLOGICAL DIAGNOSES** were: Pernicious anemia. Pulmonary congestion. Pericardial effusion. Epicardial petechiae. Fatty degeneration of the heart, liver and kidneys. Posterior sclerosis of the spinal cord. Calcified tubercles in most organs. Cholelithiasis. Anasarca.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS.

KIDNEYS: The kidneys appeared large and weighed together 550 grams and each measured 14x7x4 cm. They were firm and dark reddish-blue in color. On section there were yellowish areas running along the tubules.

Microscopically, there was granular debris within the renal tubules and Bowman's capsule. Many of the renal cells contained a brown staining pigment in abundance.

The **AORTA** showed fibrous thickening, especially near the aortic valve. Microscopically the intima showed sclerosis with the presence of crystals.

HEART: The pericardial cavity contained 300 c.c. of clear, brown-colored fluid. The heart weighed 390 grams and was dilated. The hydrostatic test showed slight mitral, and more marked tricuspid regurgitation. The valves measured as follows: M. 11.2 cm., T. 12.3 cm., A. 6.3 cm., P. 6.3 cm. The epicardium showed small petechiae. Fatty changes in the subendocardium were very pronounced, especially over the papillary muscles.

Microscopically there was extreme fatty degenerative infiltration with a moderate amount of fibroblastic proliferation. The vessels were sclerosed.

CASE 38—(59-67)

A boy of 19 years was admitted with the complaint of weakness, afternoon fever, and cough.

In his **PAST HISTORY** he had had the common childhood diseases and an occasional "cold" each winter, with little cough and sputum. About one year before admission to the hospital he had had symptoms of influenza; he had had an afternoon fever, chills, and occasional severe sweats. Since then his health had been very good. He had lost no weight and had no cough until his present illness began. His habits had always been good, his sleeping quarters for three and a half months had been in a damp basement. His best weight had been 155 pounds about five weeks before admission. He had had a thick neck for about one year.

His **PRESENT ILLNESS** had had its onset about four weeks before admission. He had had fever, weakness, anorexia, and a listless feeling all the time. He had remained in bed two days, his fever had disappeared and he had felt better. After a few days at work the fever had returned and had been usually highest in the afternoon. These symptoms had continued, and all the time he had been eating very little. About two weeks before admission he had four drenching night sweats. For the last three weeks he had gradually become dyspnoeic and there had also been a steady increase in all other symptoms, such as his weakness, fever, malaise, and anorexia. He had lost 29 pounds in weight within four weeks.

The **PHYSICAL EXAMINATION** showed a tall, very slender, cachectic patient, who looked tired. His lips, ears, and cheeks were flushed and cyanotic. His skin was warm and moist. The nose had a large amount of pus in the left side with some atrophy of the

mucosa. Eyes: Both fundi showed numerous small ill-defined yellowish-white patches, one-quarter of the disc diameter in size. The miliary tubercles in the choroid later became discrete. There was enlargement of all three lobes of the thyroid. These were soft and elastic.

The thorax was asthenic to hyposthenic in type. There was a fair expansion of the chest, slight lagging on the left; a fair Litten's phenomenon on both sides. The percussion note was rather short and high-pitched over both uppers and down to the third ribs. The breath sounds were harsh broncho-vesicular above the third ribs; occasional fine squeaks were heard in the left lower axilla. There was a positive D'Espine's sign. Dull small bubbles and occasional squeaks were heard in both bases.

The heart sounds were clear. The heart apex was visible in the 3rd and 4th interspace 8 cm., to the left. The dullness extended 3 cm. to the right and 10 cm. to the left of the midsternal line. P2 was slightly increased. The blood pressure was 105/70. The pulse was small, rapid, and regular. The temperature, pulse and respiration were respectively 104°, 120, and 26.

The LABORATORY EXAMINATIONS showed: Blood, Red blood cells 4,832,000; white blood cells 6,600; hemoglobin 80 per cent; differential count: 18 per cent lymphocytes and 80 per cent polymorphonuclears. Repeated blood cultures were negative. The Wassermann reaction was negative. The tuberculosis complement fixation test was negative. The urine was acid in reaction; specific gravity was 1.012 to 1.020, it contained no sugar, but about 25 grams of albumin per liter. Casts were occasionally present. The urine and stool cultures were negative. The phenolsuphonephthalein test showed an excretion of 60 per cent in 2 hours.

The sputum was mucopurulent, but no elastic tissue or tubercle bacilli were found. Electrocardiograms revealed a normal cardiac mechanism. The chest X-ray plate showed increase of the hilus shadow on both sides. Throughout the right lung field and upper two-thirds of the opposite lung field, faintly distributed was an immense amount of rather fine soft parenchymatous mottling, showing something of a tendency to coalescence. The thorax was long and the heart was perpendicular and apparently enlarged to the right X-ray diagnosis was pulmonary tuberculosis, miliary type.

COURSE IN THE HOSPITAL: The temperature continued high, up to 104° every afternoon and in the morning it was down to 101.8°. The respiration gradually became more difficult. The signs in the chest increased to involve both lungs with great increase in coarse crackles at the right base. The spleen was palpable at times. The condition gradually became worse and the patient was frequently delirious in the afternoon and evening. Six days after admission, the temperature had gradually increased to 105° and a corresponding increase in the pulse rate was noted. He was semi-stuporous for twelve hours. The breathing became more difficult and the pulse more rapid. He gradually became weaker and died 8 days after admission.

The CLINICAL DIAGNOSES were: General miliary tuberculosis, tuberculosis consolidation of the right lower lobe, tuberculosis choroiditis, simple goitre with the question of a tuberculous thyroiditis, atrophic rhinitis.

The PATHOLOGICAL DIAGNOSES were: Acute miliary tuberculosis of the lungs, liver, spleen, kidneys, cerebral meninges, choroid, pericardium, pleura and peritoneum, also peribronchial and mediastinal lymph glands with caseation and adhesions; chronic tuberculous perihepatitis and perisplenitis; Cystic goitre.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS.

KIDNEYS: The combined weight was 230 grams. Each measured 11.5x6x7.5 cm. with a cortex thickness of 5 mm. Numerous miliary tubercles were seen scattered throughout both kidneys. The kidneys were of a pale pink color and their capsules stripped easily, exposing in each a smooth surface which was studded with miliary tubercles.

Microscopically, the renal epithelium was swollen and granular. There were scattered throughout, miliary tubercles in considerable numbers, though they were not so numerous as in the liver, spleen, and lungs.

The HEART weighed 260 grams. Numerous caseous and calcified tubercles were seen in the epicardium, especially along the vessels. The tubercles seemed most numerous along the descending branch of the left coronary.

Microscopically, the walls of the blood vessels throughout the section were much thickened. There was also a slight general increase in connective tissue. In some parts there was an advanced fatty atrophy. Certain areas of the myocardium showed slight cloudy swelling. In the anterior papillary muscle there was a small necrotic area which was completely surrounded by many small mononuclear leucocytes, a small tubercle. The epicardium was thickened and in places contained numerous tubercles. The endocardium was thickened and in places showed a marked gathering of leucocytes beneath it.

CASE 39—(53-73)

A young Italian, aged 22, was referred into the hospital from the O. P. D. with the complaint of loose teeth, sore gums and a swollen, tender jaw.

The FAMILY and MARITAL histories were negative.

In his PAST HISTORY he had had the usual diseases of childhood without complications. (However he did not remember them specifically.) One left upper tooth had been troublesome during his army life. The filling had come out in 1921 and had never been replaced. He had an attack of severe tonsillitis in 1919 and sore throats had been troublesome, in damp weather, since this attack. He had had a cough for three months during the winter of 1918. He had no influenza or pneumonia, gonorrhea or syphilis.

He had worked as a helper in a brass foundry for eight months, then as a moulder, using phosphorous for 3 months, that is until four months before admission. While at work, he had suffered from constipation, cramps and frequent attacks of "brass chills," real

chills followed by fever and accompanied by profuse perspiration. He had worked in a planning mill for four months. He had felt weak from the time he worked in the foundry, but had had no definite symptoms.

THE PRESENT ILLNESS: The onset had been rather sudden, 15 days before admission, when he had noticed a slight pain in the jaw and the absence of the good taste, after taking a chew of tobacco. At lunch on the same day, he had felt a severe pain in the left side of the lower jaw, on biting into an apple, there was no swelling at this time. Four days later a dentist evacuated pus from beneath the painful lower incisor tooth. It was considered inadvisable to pull the tooth. Within two days, however the tooth and an adjacent one were lifted out. The lip began to swell. It was incised by a surgeon, the abscess drained pus and was irrigated. After the swelling of the lip receded, the jaw began to swell. The gums became soft and spongy and all the lower teeth became loose. The pain in the jaw was constant with intermittent acute exaggeration. He suffered from general weakness and malaise.

The **PHYSICAL EXAMINATION** showed a marked pallor and a pasty, yellow tinted clammy skin. Acromegalic features were present. The enlarged lower jaw was very tender. The maxilla was thickened and the lips were swollen. The mouth could be opened only 2 cm. The mucous membranes were very pale. The lower gums were spongy, swollen and fissured. The lower four incisors, the canine and the bicuspid on the left were missing, and the alveolar sockets discharged pus. The remaining lower teeth were loose. There was no marked infiltration in the floor of the mouth. A general glandular enlargement was present in the neck glands.

CHEST: The lungs were negative.

HEART: A distinctly increased impulse was felt over a wide area. No enlargement was made out. The heart sounds were of good quality. A very distinct presystolic gallop rhythm was noted at the apex. P2 was loud and slightly accentuated. The blood pressure was 130/80. The heart rate was 100 and regular. The arteries were not thickened.

ABDOMEN: Hard, sharp border of the spleen was felt $2\frac{1}{2}$ cm. below the costal margin. The liver was not palpable.

EXTREMITIES: The reflexes were slightly sluggish. The hands and feet were thickly calloused.

The **LABORATORY EXAMINATIONS** showed: The urine was acid 1009-1018 in specific gravity and contained a trace of albumin, rare hyalin casts, red blood cells and white blood cells. The 'phtalein excretion was 45 per cent in the first hour and 20 per cent in the second—a total of 65 per cent. The blood was that of a leukemia with a severe secondary anemia. Hemoglobin 30 per cent; red blood cells 1,950,000 and white blood cells 96,000. The differential count showed lymphocytes 55 per cent, endotheliocytes 30 per cent, polymorphonuclear neutrophils 15 per cent, and polymorphonuclear basophils 5 per cent. The blood Wasserman was negative. Blood cultures were negative. The electrocardiogram showed T. III slightly inverted and right ventricular preponderance.

COURSE IN THE HOSPITAL: The T. P. R. averaged T. 39° - 40° . P. 90-130, and R. 22-48. Transfusions caused a doubling of the red cell count, while the leucocytes dropped to 16,000. Bronchopneumonia developed in the right lower lobe of the lung, also a pleurisy with effusion at the right base. He lost ground rapidly after the onset of the lung complications and died exactly one month after admission.

The **CLINICAL DIAGNOSES** were: Necrosis of the inferior maxilla. Acute, cervical and submental lymphadenitis. Lobar pneumonia in the right lower lobe and bronchopneumonia in the right and left. Acute serofibrinous pleurisy, Leukemia? (Lymphatic? Myelogenous?). Oedema of brain. Secondary anemia. Septicemia. Acute gingivitis. Mitral stenosis (?) Acute general emacipation.

The **PATHOLOGICAL DIAGNOSES** were: Osteomyelitis of the inferior maxilla with necrosis of bone and suppuration of neighboring soft tissue. Pustules of skin of neck and anterior chest. Suppurative pneumonia with abscesses of right lung and gangrene. Bronchitis and bronchopneumonia of the left lung. Empyema of right chest with localized pus pocket. Hyperplasia of the mesenteric, right axillary and epitrochlear lymph nodes. Hyperplasia of the bone marrow. Early aortic sclerosis. Calcified and caseous tuberculous peribronchial lymph nodes. Healed operative scar of left epitrochlear region. Adhesions about the appendix. Infected operative wound of left axilla. Chronic orchitis. Unidentified gram positive bacilli were cultured from the right chest, pleura, bronchi and lungs. Non-hemolytic Streptococci and Staphylococcus pyogenes albus were cultured from the Mediastinal abscess.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS.

The kidneys together weighed 430 grams and each measured $13 \times 4.5 \times 6.5$ cm. The capsule was adherent and left several injected areas on the surface. There were small gray-white nodules up to 3 mm. in diameter on the surface and these were also visible on section. The cortex was of normal thickness, but the striations were not marked. The glomeruli showed up distinctly injected.

Microscopically, in one area amongst cortical tubules there was a little cellular accumulation of large mononuclears and a few polymorphonuclears. The cytoplasm of the tubules was granular and appeared swollen.

The **AORTA** showed a few raised patches throughout. Microscopically, there was only a slight intimal thickening.

HEART: The pericardial cavity contained a slight excess of clear straw-colored fluids. The heart weighed 230 grams. The valve rings appeared normal in size. The mitral measured 12.5 cm. and was slightly thickened, the tricuspid 13 cm., the pulmonary 6.5 cm., the aortic 6 cm. No marked abnormalities of valves were found. The heart muscle was flabby.

Microscopically, there was a diffuse infiltration of mononuclear leucocytes throughout the myocardium. At several places in the epicardial fat there was a marked leucocytic infiltration. Many of the leucocytes were of the plasma-cell type. There was also some fibroblastic proliferation. Some of the muscle fibers showed advanced cloudy swelling.

CASE 40—(4-4)

A boy, aged 13, was admitted with the complaint of pain in the right side of the abdomen.

His FAMILY HISTORY was negative.

His PAST HISTORY was likewise negative.

His PRESENT ILLNESS began 4 or 5 weeks before admission to the hospital with enlargement of the cervical lymphatic glands. Ten days before admission to the hospital the child had complained of pain in the abdomen and nausea. Two days later he had begun to vomit after each feeding. The urine was examined by the attending physician who diagnosed "kidney trouble". The patient had been given daily cathartics and enemata, and no other treatment. His weakness had rapidly increased. He had vomited blood two days before admission.

On admission to the hospital the PHYSICAL EXAMINATION revealed a well developed boy complaining of abdominal pain. He was apparently completely prosterated. The breath was extremely foul. The mouth was dry, and showed some ulcerated patches. The tonsils were large and inflamed, and covered with a small amount of exudate. There were numerous petechial spots over the lower chest and upper abdomen and on the back. All superficial lymphatic glands were moderately enlarged, discrete, firm and not tender. The eyes were normal except for an extensive subconjunctival hemorrhage in the left eye.

The chest was negative. The lungs were clear. The heart was not enlarged. The first sound at the apex was harsh. There was no murmur. The rhythm was regular and the rate 72 per minute.

The abdomen was at the level of the costal margin. There was slight rigidity, more marked on the right. A definite mass the size of a small orange was felt in the right lower quadrant. The whole abdomen was rather doughy. The liver and spleen were not felt. The temperature was 100, respirations 24, and pulse 90.

The LABORATORY FINDINGS: The blood showed W. B. C. 17,000. The urine was turbid, contained a large amount of albumin, some casts, numerous R. B. C. and W. B. C. Non-protein nitrogen in the blood was 128 mgm. per 100 c.c. The blood pressure was 110/75. The electrocardiogram showed a low QRS group in lead III.

COURSE IN THE HOSPITAL: The patient continued to vomit almost everything taken. The vomitus contained a considerable amount of altered blood on one or two occasions. The petechial spots over the body became more numerous, and hemorrhages from the gums were noted together with the development of a sub-conjunctival hemorrhage in the right eye. There was severe abdominal pain at intervals, referred chiefly to the right lower quadrant, with extreme tenderness in the same region. Progressive prostration was evident. Numerous rales were heard in the lungs the day preceeding death. The temperature remained below 100° during five day's stay in the hospital. A blood count done several hours after the one made on admission, showed 80,000 W. B. C. A differential count showed polymorphonuclears 3 per cent; large lymphocytes 69 per cent; small lymphocytes 28 per cent; and numerous normoblasts. The hemoglobin was 75 per cent, the red blood cells numbered 3,500,000. Subsequent white blood counts varied from 80,000 to 104,000. The differential count was essentially the same as previously. Treatment consisted of water by rectal drip, liquid diet and morphine for the control of abdominal pain.

The CLINICAL DIAGNOSES were: Acute lymphatic leukemia. Bronchopneumonia.

The PATHOLOGICAL DIAGNOSES were: Lymphoid leukemia involving the lymphoid tissue throughout the body and infiltrating all organs. Multiple hemorrhages into the serosa of the peritoneal cavity, the skin and conjunctiva. Chronic localized fibrous pleurisy.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS.

KIDNEYS: The kidneys were large and pale, with the appearance of the typical large white kidney. The left weighed 280 grams and the right weighed 270 grams. The cortex measured 1 cm. in its thinnest part. The medulla was pale, but still it was darker than the cortex.

Microscopically, the vessels were gorged with leukemic lymphocytes, which in places infiltrated the myocardium to a considerable extent. The myocardial cells showed cloudy swelling, simple necrosis and fatty degenerative infiltration. The subepicardial fat was increased and also infiltrated with the lymphocytes.

CASE 41—(27-99)

A bachelor, aged 70, was admitted with a post-operative perineal fistula.

His PAST HISTORY was irrelevant. He denied ever having had any venereal disease.

The PRESENT ILLNESS had begun 4 years before admission, when he had an obstruction and retention of the urine. He was told at the time that he had a large prostate which was causing obstruction. Operation was advised. The prostate was removed by the perineal route. Since the operation he had had a persistent fistula. He entered the hospital for repair of the fistula.

The PHYSICAL EXAMINATION showed a perforated nasal septum. Generalized arteriosclerosis. A hydrocele and a rectovesicular fistula.

The lung apices showed the signs of consolidation.

The heart was not enlarged. The rhythm was regular. There were no thrills or shocks. The blood pressure was 150/67. No cyanosis, dyspnoea or engorged neck veins were present.

The abdomen was negative. The extremities were emaciated.

The LABORATORY EXAMINATIONS showed: The urine contained a large amount of albumin. The phenolsulphonphthalein test was unsatisfactory.

The blood counts and hemoglobin estimations gave evidence of a slight anemia and a moderate leucocytosis. The blood Wassermann was "four plus" and the blood non-protein nitrogen was 36 mg. per 100 c.c.

COURSE IN THE HOSPITAL: The patient developed pneumonia following the repair of the fistulae. A massive dose of digitalis was administered without much effect. The electrocardiogram showed single and runs of auricular extrasystoles. The pneumonic process failed to be affected by treatment and the patient died within a week, that was three and one-half months after admission.

The **CLINICAL DIAGNOSES** were: Fistula in ano and recto-urethral and rectovesical fistulae. Chronic cystitis. Enlargement of the prostate. Hydrocele of the right tunica vaginalis. General arteriosclerosis. Syphilis. Perforated nasal septum. Phlebitis of the left saphenous vein. Auricular extrasystoles. Lobar pneumonia of the right lower lobe.

The **PATHOLOGICAL DIAGNOSES** were: Tuberculous pneumonia of the right lower lobe with cavitation. Old pleural adhesions. Bronchopneumonia with purulent bronchitis. Emphysema. Pulmonary oedema. Pleura effusion. Suprapubic prostatectomy. Membranous cystitis. Chronic interstitial nephritis. Nephrolithiasis. Fatty degeneration of the liver. Chronic perisplinitis. Cholelithiasis. Dilatation of the aorta with relative valve insufficiency.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS.

KIDNEYS: The right kidney weighed 150 grams and measured 10x5.5x2.5 cms.; the left weighed 200 grams and measured 12x5.5x3 cms. The cortex of each measured 7 mm. The capsules stripped with difficulty, tearing the kidney in a number of places and leaving a scarred surface. The tissue was pale pinkish gray in color and the cortical striations were very irregular and the kidney substance scarred. In the left kidney there were a number of small yellow soft stones, some of which were in the pelvis, while some were still in the tubules.

Microscopically, the kidneys showed marked granular changes in the cells of the collecting tubules, many of these cells had no nuclei and the tubules contained much debris. Bowman's capsules were intact. The blood vessels had thickened walls. There was no great amount of scarring, but there was a small amount in a few areas.

The **AORTA** was only slightly elastic. There was definite wrinkling of the wall and the intima but no large atheromatous patches were noted. The root was somewhat dilated, but insufficiency of the valve was very questionable.

The **HEART** weighed 295 grams. The muscle was of good color. The valves appeared normal and measured as follows: Aortic 8 cm., mitral 10 cm., pulmonary 7 cm., tricuspid 12 cm.

Microscopically, there was a moderate increase in connective tissue throughout. The myocardium showed also a diffuse mononuclear infiltration. The endocardium was thicker than normal in some places. The epicardium and subepicardium fat and adjacent myocardium showed many colonies of bacteria, which were surrounded by leucocytes. The muscle fibers for the most part were smaller than normal and showed some cloudy swelling.

CASE 42—(28-98)

A widow, aged 63, was admitted with the complaints of pain in the right lower quadrant of the abdomen; vomiting of foul, brown matter; frequent, painful urination; and constipation.

Her **FAMILY HISTORY** was not especially significant.

In her **PAST HISTORY** she had had diphtheria at 3 years of age; scarlet fever at 8; a growth removed from the urethra in 1907; pneumonia in 1910 and 1914; enlarged, painful joints for three or four years, especially in winter, and slight swelling of the ankles during the past year.

The **PRESENT ILLNESS** had begun ten days before admission with a constant sharp pain in the right lower quadrant of the abdomen. She had had a temperature of 103° on the third day. She had begun to vomit on the fifth day. The bowels had moved slightly each day with a simple enema. On the seventh day the vomitus had been dark-brown, foul-smelling, and considerable in amount. The pain then had become more severe in the region of the umbilicus. She had had burning and frequency of urination for five or six days. Turpentine applied to the abdomen had caused erythema and vesiculation.

The **PHYSICAL EXAMINATION** showed an obese woman with a generalized abdominal tenderness, which was more marked below the umbilicus. There was no rigidity. The temperature was 100.6°. The lungs and the heart were negative. The blood pressure was 140/70.

The **LABORATORY EXAMINATIONS** showed: Blood, Hgb. 70 per cent; r. b. c. 4,832,000 per cu. mm.; w. b. c. 22,000 per cu. mm.; the differential count showed 90 per cent polymorphonuclears. The urine contained a trace of albumin and many hyaline and granular casts. The stools showed a positive benzidine reaction. The Wassermann reaction was negative. The blood non-protein nitrogen was 139 mg. per 100 c.c. The electrocardiogram showed left ventricular preponderance.

COURSE IN HOSPITAL: There was an irregular slight rise in temperature, rarely reaching 101°. The pulse was 100. The respirations were 22. The leucocytes persisted at slightly over 20,000. The blood N. P. N. remained at 135 mg. and 111 mg. per 100 c.c. The patient became increasingly more stuporous.

A gynecological consultant found the body of the uterus enlarged and fixed and the upper boundary was not made out because of abdominal tenderness. A diagnosis of malignancy of the body of the uterus with peritoneal metastases was made. The patient gradually grew worse. A pyoderma and bed sores developed. The blood pressure dropped to 110/65, the stupor increased, a slight cough appeared and rales were heard at the left base. Cold clammy sweats became disturbing. The patient became more and more toxic and died one month after admission.

The **CLINICAL DIAGNOSES** were: Intestinal obstruction, uremia, chronic interstitial nephritis, chronic myocarditis, bronchopneumonia, carcinoma of the uterus.

The **PATHOLOGICAL DIAGNOSES** were: Appendiceal abscess, generalized subacute peritonitis with multiple pus pockets in the mesentery and omentum, localized peritoneal

abscess with fecal concretion, old organized adhesions about the cecum, anthracosis of the lung, pleural adhesions, bronchopneumonia, cholelithiasis, multilocular cyst of the pancreas, healed tuberculosis of the spleen, mild acute cystitis, endometritis, retroversion of the uterus. The bacteriological examinations showed *B. coli* in the heart's blood, in the abscess, and in the kidneys.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS.

KIDNEYS: The right kidney weighed 125 grams and measured 12x5.5x3 cm. and the cortex averaged 7 mm. The left kidney weighed 130 grams and measured the same as the right. The organs were pinkish-gray in color. The capsules stripped with ease. On section the cortical striation were distant and there was no congestion or other abnormality to be made out.

Microscopically, the glomeruli appeared normal with Bowman's capsule intact. There was a small amount of hyalinlike change in the vessels at their points of entrance into the glomerular tufts. These areas of homogeneous material did not give the metachromatic reactions for amyloid. The epithelial cells lining the tubules were somewhat flattened and the lumina of the tubules contained considerable debris. The tissue spaces were distended and occasionally one of these spaces was found which contained debris similar to that in the tubules. There was moderate increase in connective tissue between the tubules and an occasional glomerulus was sclerotic.

The HEART weighed 248 grams and appeared normal in size. The valves were competent and showed no pathological changes. The valve rings measured as follows: mitral 9 cm., aortic 7 cm., tricuspid 19 cm., and pulmonary 6.5 cm.

Microscopically, there was a diffuse connective tissue infiltration. The myocardium showed considerable fragmentation of the muscle cells. The vessels were sclerotic and congested and some contained bacterial colonies. The endocardium was thickened and fibrosed, with a subendocardial myocardium showing fatty degenerative infiltration.

CASE 43—(42-84)

A married man, aged 44, entered the hospital complaining chiefly of headache and high fever.

His PAST HISTORY and MARITAL HISTORY were irrelevant.

The PRESENT ILLNESS had begun with a history of furunculosis, and a pimple on the nose about two weeks before admission. Eight days before admission he had had a chill. Two days later there had developed a cold "in the head" and fever. Three days after the first chill he had begun to have severe headaches, chills and fever with a temperature of from 100° to 104°. On admission he had a headache over the left eye and left side of the head. The temperature was 101.5°, the pulse was 86, the respirations were 36.

The PHYSICAL EXAMINATION showed discoloration and ptosis of the left upper eyelid, double vision, weakness of the internal and external recti on the left, with possibly the 4th cranial nerve also involved. There was exophthalmus on the left with chemosis and injection of the conjunctiva. The corneal reflexes were equal. The pupils were unequal, the left being larger than the right. The left did not react to light, while the right reacted slightly. The discs on ophthalmoscopic examination showed; in the right eye, disc outline blurred, general hyperemia, the veins dark and engorged and slightly tortuous; in the left eye, disc outline blurred in the inferior and nasal margins, hyperemia, and the veins dark and engorged. There was some edema to the inner side of the disc extending into the retina. The relation of the width of the veins to the arteries was more than two to one.

There was no objective sensory findings. The reflexes were very active. No clonus or pathological toe signs were present. Some resistance in the neck muscles was present, but no definite rigidity.

COURSE IN THE HOSPITAL: The next morning there was marked exophthalmus on the left and beginning on the right. The chemosis and swelling of the lids was increasing; the temperature was 105°. Ophthalmoscopic examination in addition to the above findings showed some edema and exudate in both eyes. A lumbar puncture yielded cloudy fluid, the pressure of which was increased. The cells numbered 2,300, with 90 per cent polymorphonuclears. The globulin and albumin tests were strongly positive. No organisms were found in the smear. The lumbar puncture on the following day showed 2,600 cells and no organisms. The temperature ranged between 104° and 105°; pulse 84, respiration rate was 30. There was marked bilateral exophthalmus and discoloration of the upper lids and congestion of the temporal veins.

On the third day in the hospital marked spasm of the neck muscles was noted; no pathological toe signs, clonus, or Kernig were elicited. The T. P. R. chart showed: temperature 103° to 105.8°, pulse 100, respirations 38. The physical signs were as above except that the patient was irrational and stuporous, with the respiration noisy and Cheyne-Stokes in type. The spinal fluid contained 7,900 cells per cu. mm., 88 per cent of which were polymorphonuclears.

On the fourth day the eye signs were increased, discoloration, exophthalmus, and swelling of the discs were more marked. The T. P. R. was as above. The stupor was greater.

On the fifth day the patient was comatose and exhibited marked Cheyne-Stokes respiration. There were involuntary defecations and urination. Lumbar puncture showed 800 cells, 74 per cent of which were polymorphonuclears.

On the sixth day a greenish-yellow discoloration of the face, eyelids and temporal region was present. Cheyne-Stokes respiration continued. The pulse was weak and thready with a rate of 140 to 180. Many rhonchi and bubbling rales were audible. The patient rapidly lost ground and died in the early hours of the seventh day in the hospital.

LABORATORY FINDINGS: The urine contained a slight amount of albumin, with hyaline and granular casts, which finding increased. The blood picture was normal. The blood and spinal fluid Wassermann reactions were negative. The blood and spinal fluid cultures were negative.

The CLINICAL DIAGNOSES were: Bilateral cavernous sinus thrombosis, cerebrospinal meningitis.

The PATHOLOGICAL DIAGNOSES were: Septic thrombosis of the cavernous sinuses, purulent streptothrix meningitis, septic streptothrix embolism of the lungs, edema and ecchymoses of the conjunctiva; streptothrix abscess involving the hypophysis and sella turcica.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS:

KIDNEYS: The right kidney weighed 185 grams and the left 160 grams. The cortex measured 6 mm. at its thinnest point in each. There were localized thickenings in the capsule, but these did not appear to extend into the kidney substance. In the right kidney there was one rather firm, whitish area in the cortex and two similar areas in the medulla. These areas did not contain pus and did not resemble localized fibrosis. Microscopically, there were a few sclerotic glomeruli in areas in the cortex near the capsule, which were infiltrated with lymphocytes. In some parts of the kidney there was a small amount of perivascular infiltration with lymphocytic elements. Some of the tubules contained hyaline casts. In one small vessel there was a fibrinous clot and numerous leucocytes, but no organisms could be identified.

The AORTA was elastic and microscopically, there were moderate atheromata, with plasma cells and large mononuclears in the intima.

The HEART weighed 295 grams and was not abnormal externally. The mitral, tricuspid, aortic, and pulmonary valves were negative.

Microscopically, there was a diffuse mononuclear leucocytic infiltration throughout the myocardium. The muscle cells showed cloudy swelling. There was slight fibroblastic proliferation in the myocardium. The endocardium was slightly thicker than normal. The subpericardial fat was increased in amount.

CASE 44—(55-71)

An Irish clerk, aged 35, was admitted with the complaints of weakness, nausea and vomiting and pains all over the abdomen, in the arms and legs, sallow color, and dyspnoea.

His PAST HISTORY was irrelevant. He had had gonorrhea one year before his chief symptoms had begun.

His PRESENT ILLNESS had begun one year before his last admission. During this year he had been in the hospital four times for periods of one to four months. In spite of transfusions and slight remissions there had been a gradual increase in symptoms. A chronic prostatitis had lighted up and had begun discharging. Vomiting of yellow fluid before breakfast, diarrhoea, numbness and tingling of the fingers had become troublesome. He had been feeling fairly good with the exception of the pains in legs, until about a month before his last admission. The week before admission he had become very weak and unable to walk, with dyspnoea and severe pains over the entire body. He had complained of severe sharp pains over the abdomen on admission and had been unable to retain any food except small sips of liquid.

The PHYSICAL EXAMINATION showed a very weak, sallow individual who was considerably disturbed by occasional vomiting of dark greenish fluid. His expression was one of anxiety with anxious staring eyes. His face was yellow and waxy and the rest of his skin very sallow. The sclerae were lemon yellow in color.

The lungs were hyperresonant. The breath sounds were increased at the right apex, where also spoken and whispered voice sounds were slightly increased.

The heart was apparently not enlarged. A soft systolic murmur was heard at the apex. The heart sounds were distant. The blood pressure was 110/60.

The abdomen was tender throughout. The spleen was barely felt. The right K. K. and A. K. were sluggish. There was a positive Babinski's and Gordon's signs on the left. The Romberg was positive with a tendency to falling to the left. There was ataxia on the heel to knee test. There was no superficial sensory changes.

The LABORATORY EXAMINATIONS showed: The blood: R. B. C. 1,296,000; W. B. C. 1,400; Hemoglobin 22 per cent. The differential count and smear showed typical pernicious anemia changes, there were 4 per cent normoblasts. Fractional gastric analysis showed an achlorhydria. The urine showed a trace of albumin and a few granular casts. The Wassermann was negative. Electrocardiograms showed T waves inverted in leads II and III.

COURSE IN THE HOSPITAL: On admission the temperature was 103.2°. The patient suffered frequent vomiting of greenish fluid and was irrational at times. The temperature followed a remittant course, the respiration gradually became more rapid and difficult. The delirium increased. The vomiting ceased and the patient ingested much fluid. He was given two blood transfusions of 800 c. c. and 650 c. c. respectively with no improvement resulting. The course of his disease was steadily downward because of an intercurrent bronchopneumonia. The blood improved showing R. B. C. 2,984,000; W. B. C. 2,850 and Hemoglobin 49 per cent. In spite of this, the respiration steadily became very difficult and the pulse very weak. He died one week after admission.

The CLINICAL DIAGNOSES were: Pernicious anemia. Combined sclerosis. Chronic prostatitis. Chronic pulmonary tuberculosis. Edema of the lungs. Chronic passive congestion of the kidneys. Bronchopneumonia.

The PATHOLOGICAL DIAGNOSES were: Pernicious anemia. Hyperplasia of the spleen and bone marrow. Infarct in left lower lobe of the lung. Bronchopneumonia. Acute cardiac dilatation. Cloudy swelling of the kidneys. Right hydronephrosis. Chronic passive congestion of the stomach and lungs. Perisplenitis, perihepatitis and fibrous peritonitis. Acute focal enteritis. Chronic enteritis. Focal and healed pulmonary and gland tuberculosis.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS.

The KIDNEYS weighed 565 grams together and each measured 12x5x4 cm., and appeared

swollen and pale. Microscopically, the kidney epithelium was swollen and granular. The blood vessel walls were thickened.

The HEART was flabby and fatty otherwise there was no abnormality of the valves. No other lesions were found in the muscle or vessels.

Microscopically, there was a slight mononuclear leucocytic infiltration in the subepicardial connective tissue. There was a slight increase in connective tissue, and some mild cloudy swelling in the myocardium. The endocardium was thickened, but showed no conspicuous infiltration of cells. The subendocardial myocardium contained some areas of cells showing a mild fatty degenerative infiltration.

CASE 45—(38-88)

A farmer, aged 52, was admitted complaining of weakness, loss of weight, shortness of breath and pain over the heart.

The FAMILY HISTORY and MARITAL HISTORY were unimportant.

The PAST HISTORY was negative except for a severe attack of malaria at the age of 27.

The PRESENT ILLNESS had begun quite insidiously. One year before admission he had become too weak to do his work. Three months later, besides tiring easily with any physical effort, he had also noted a definite shortness of breath and palpitation. He had had to sit down and rest after any exertion, such as climbing one flight of stairs.

One month before admission he had felt a soreness under the last rib on the left side. The discomfort had not been a distinct pain, but an ache, which had been exaggerated by the indigestion of food. He had had no nausea or vomiting and his appetite had been fairly good. Hemorrhoidal tags had been removed just before admission, but an exaggeration of the local pain and the general symptoms had resulted.

The PHYSICAL EXAMINATION revealed pallor, emaciation and cachexia. The hearing was impaired bilaterally. The mucous membranes were pale. The tongue was red and slightly atrophied.

The lungs were clear and resonant throughout.

The heart was not enlarged. The cardiac impulse was in the 4th intercostal space 9.5 cm. to the left of the midsternal line. No thrills or shocks were felt. The cardiac dullness extended 4 cm. to the right and 9.5 cm. to the left of the midsternal line. The rhythm was regular. The sounds were distant. The first sound at the apex was replaced by a blowing systolic murmur which was transmitted into the axilla and toward the sternum. The second heart sound along the left border of the sternum and in the aortic area was impure. The radial arteries were sclerosed and beaded; the brachials were thickened and the temporals were tortuous. The pulse was full but quick. The blood pressure was 95/50.

The abdomen contained a mass that extended 4.5 cm. below the costal margin in the nipple line. The mass was firm and flat and descended on deep inspiration. There was tenderness in the epigastrium and in the mass. The liver was questionably palpable.

Enlarged glands were found in the axilla, especially the left and in the inguinal regions.

There was an irregular fever often reaching 104.4°.

The LABORATORY EXAMINATIONS showed: Blood: Hemoglobin 30 per cent, red blood cells 2,100,000, white blood cells 10,600. Differential Count, polymorphonuclear neutrophils 72 per cent, endotheliocytes 6 per cent, lymphocytes 7 per cent, and myelocytes 15 per cent. The Wassermann reaction was negative. Blood cultures remained sterile.

The urine was negative. The 'phthalein excretion was 30 per cent in the first hour and 25 per cent in the second hour.

The stools gave a strongly positive reaction for occult blood.

The gastric analyses revealed a persistent achlorhydria.

Gastro-intestinal X-ray studies showed a mass involving the upper two-thirds of the stomach.

COURSE IN THE HOSPITAL: The patient was operated, and a large round tumor was felt through the anterior wall of the stomach. A similar tumor was found on the posterior wall and along the greater curvature. Both masses were adherent posteriorly. The peritoneum was oedematous and a bloody fluid containing fibrin flakes was present, suggesting the presence of an early peritonitis. The abdomen was closed without drainage. The patient died three days later, that is, fifteen days after admission.

The CLINICAL DIAGNOSES were: Carcinoma of the stomach. Severe simple anemia. General arteriosclerosis. Internal hemorrhoids. Acute general peritonitis.

The PATHOLOGICAL DIAGNOSES were: Adeno-carcinoma of the stomach with an old perforation. Acute purulent general peritonitis. Acute splenic tumor. Cloudy swelling of the kidneys. Chronic fibrous pleurisy. Focal calcified tuberculosis. Diverticulum of the gall bladder. Chronic interstitial pancreatitis. The peritoneal cultures contained hemolytic streptococci.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS.

The KIDNEYS weighed 225 grams and each measured 11x6.5x2.5 cm. with an average cortex thickness of 8 mm. The capsules stripped easily from both, exposing in each case a pale red surface upon which congested capillaries were conspicuous. The surface was smooth. The normal kidney markings could be distinguished on the cut surface. The cortical zones were swollen and pale yellow radiating lines could be seen in the pyramids and to a less extent yellow lines could be seen in the region of the collecting tubules.

Microscopically, there was a proliferation of the intima so advanced in some of the smaller branches of arcuate arteries as to nearly obliterate the lumen. Dilated tubules contained hyalin plugs here and there. The tubular epithelium was swollen and granular.

The HEART was about normal in size. The muscle appeared pale, flabby and poorly contracted. The valves all appeared thickened and increased in size. The hydrostatic test showed regurgitation through the mitral and tricuspid valves.

Microscopically, there were many small foci of polymorphonuclear leucocytes throughout.

With this infiltration there was a fibroblastic proliferation. The subepicardial fat was only slightly increased. There were some local thickenings of the endocardium with a slight leucocytic infiltration. The walls of the blood vessels were only slightly thickened. The myocardial cells were slightly diminished in size.

CASE 46—(17-174)

A married man, aged 52, was admitted with the complaints of severe headache and pain in the right side of the face.

The FAMILY HISTORY and MARITAL HISTORY were not significant.

In his PAST HISTORY he had had mumps, measles, pertussis and scarlet fever in infancy with no complications. He had had malaria at 21 and gonorrhea at 22, 25, and 27. He had apparently not had syphilis. He had had pleurisy at 47. He had consumed two glasses of beer, an occasional whiskey, three cups of coffee and one pipeful of tobacco per day.

The PRESENT ILLNESS had begun about two months before admission. He had had influenza and had begun to have severe occipital headaches extending over the vertex to the forehead and down the right side of the face. Attacks had begun each evening and had lasted all night. Except for weakness he had felt good each morning. For two years he had had severe headaches once or twice per week. These had lasted only a few hours and had been promptly entirely relieved. He had had to vomit occasionally but had not vomited in the last two years. He had had no trouble with his eyes. Hearing in the right ear had been gradually lost within two months of admission.

The PHYSICAL EXAMINATION revealed normal pupils with prompt reaction to light and accommodation. There was slight ptosis of the left upper eye lid. Slight lateral and anticlockwise nystagmus was found. The hearing was practically lost in the right ear. The right side of the face and the right cornea were hyperalgesic. The pharynx was injected. The neck was negative.

The lungs were clear and resonant throughout.

The heart was not enlarged. The cardiac dullness extended 3 cm. to the right and 9 cm. to the left of the midsternal line. The first sound was harsh in the mitral and tricuspid areas. The pulmonary and aortic second sounds were clear and loud. The heart rate was 92 per minute and the rhythm was regular. The blood pressure was 142/90 and 128/80.

ABDOMEN. The liver, spleen and kidneys were not palpable. There was a right inguinal hernia. The extremities showed no abnormalities.

The LABORATORY EXAMINATIONS showed: The urine was negative. The 'phthalain output was 22 per cent the first hour and 32 per cent the second hour. The blood examination revealed 85 per cent hemoglobin, 4,240,000 red blood cells and 8,250 white blood cells. An X-ray plate showed a large right internal auditory meatus. The electrocardiogram showed a questionable right ventricular preponderance.

COURSE IN THE HOSPITAL: The patient died on the operating table of respiratory failure before the dura mater was opened.

The CLINICAL DIAGNOSES were: Tumor of the eighth cranial nerve. Right inguinal hernia.

The PATHOLOGICAL DIAGNOSES were: Glioma of the eighth cranial nerve. Craniotomy. Chronic fibrous and calcified focal and apical pulmonary and fibrous tuberculosis and tuberculous lymphadenitis. Disseminated encapsulated tuberculosis of the kidney and spleen. Chronic perisplenitis. Chronic nephritis. Hypertrophy of the prostate. Chronic colitis with lymphoid hyperplasia.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS.

KIDNEYS: The right kidney weighed 140 grams and measured 12.5x6x3.5 cm. The left weighed 140 grams and measured 11x7x3 cm. The cortex measured 6 mm. in each kidney. The capsules stripped with some difficulty because of friability. There were no adhesions. The surface was smooth, deep red in color and normal markings could be distinctly made out on the cut surface. There was near the surface of the lower pole of the left kidney a cyst which contained clear fluid and measured 1.5 cm. in diameter. It was elevated somewhat above the surface. Near the upper pole of the ventral surface of the of the same kidney there was a small yellow caseous focus about 2 mm in diameter. This extended into the kidney substance and was not elevated above the surface.

Microscopically, the kidney showed advanced parenchymatous degeneration of the tubular epithelium. There were diffuse scars in the region of the collecting tubules. Many tubules contained hyaline plugs, others were dilated and partially filled with desquamated epithelial cells.

The AORTA showed yellowish plaques in the ascending portion of the arch.

The HEART appeared slightly enlarged. It was moderately distended with blood and there were clots in all the chambers and the arteries. The valves appeared normal.

Microscopically, the fibers of the myocardium were swollen, granular and fragmented. The cross striations, however, were still evident in most of them. There was an advanced extensive fatty atrophy in some parts of the myocardium. The subepicardial fat was increased in amount. There was also more or less generalized marked cloudy swelling. The connective tissue was very much increased in amount.

CASE 47—(14-14)

A female infant, aged 7 months, was brought into the hospital because of grunting, (like an old woman) and an inability to gain weight.

The FAMILY HISTORY was significant in that the father had had an intermittent discharge and had admitted having had gonorrhea before the child was born, but denied

syphilitic infection or symptoms of it. There was one other child three years old living and well. The mother had had no miscarriages.

The PAST HISTORY and the present illness must be considered together. The infant had been a seven months babe, according to the midwife, but had weighed seven pounds. Snuffles had begun at the age of one month. Breast feeding had been carried out until the fifth month, when the mother's breasts dried up. Cow's milk had then been tried, but the baby would not take it, and Eagle Brand Condensed Milk had been substituted. The infant had failed to gain weight. At the age of two months the babe had been seen in the O. P. D. with snuffles, sore mouth, malnutrition, large spleen, scaling palms and soles and opisthotonus. Lumbar puncture had shown a cloudy fluid with 800 mononuclear cells. The patient had not been seen after that until admission, though the symptoms had slowly but definitely progressed.

The PHYSICAL EXAMINATION showed a poorly nourished and poorly developed infant assuming an opisthotonus position. The neck was stiff and bending it was painful. Cheyne-Stokes respiration was noted. The head showed evidence of a mild hydrocephalus, the circumference measured 40 cm. The fontanelles were open, the anterior was especially widely open with a spreading of the sutures at the angles; the posterior was one cm. in diameter. The anterior fontanelle was bulging and tense. The skin over the head was stretched and shining. The veins of the scalp were markedly dilated. The eyes protruded. The fundi showed chorio-retinitis and choked discs.

The lungs were clear. The heart was not enlarged. The action was rapid but the sounds were of fairly good quality. No murmurs were heard. The axillary veins were dilated.

The abdomen was distended and presented a slight umbilical hernia. The liver was 2 cm. below the costal margin. The spleen was just palpable. Kernig's and Brudzinski's signs were positive. There was a general glandular enlargement noted.

LABORATORY DATA: The blood Wassermann reaction was "four plus." The spinal fluid was cloudy and contained 800 cells. The mother's blood Wassermann reaction was "four plus." The urine contained a slight amount of albumin and many pus cells. The blood showed 80 per cent hemoglobin and 11,300 leucocytes.

Electrocardiograms showed, low Q. R. S. groups in leads I, and III and the T wave inverted in lead III.

COURSE IN THE HOSPITAL: The babe was given mercurial inunctions and five .55 dg. doses of Salvarsan. The patient gained weight on adjusted feedings. One month after admission erysipelas developed over the head, eyes and face. The temperature rose to 104 degrees and remained there, and the patient died three days later.

The CLINICAL DIAGNOSES were: Congenital syphilis. Syphilis of the cerebrospinal meninges. Erysipelas.

The PATHOLOGICAL DIAGNOSES were: Erysipelas with extension into the mediastinum. Acute mediastinal lymphadenitis. Acute interstitial and glomerular nephritis. Acute splenic tumor. Hyperplasia of the splenic corpuscles and the mesenteric lymph nodes. Chronic perisplenitis with adhesions. Chronic meningitis. Bronchopneumonia. Streptococcus hemolyticus in the lung, heart blood and skin.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS.

KIDNEYS: The right kidney weighed 32 grams and measured 6x3.5x2.2 cm. and the left weighed 32.5 grams and measured 7.5x3.5x2 cm. The cortex of each measured 4 mm. The kidneys were lobulated. The capsules stripped with ease. They had a grayish mottled appearance. The cut surfaces had pale swollen appearance and there was very little bleeding. Microscopically, there were areas of round cell infiltration in the cortex. Few polymorphonuclear leucocytes could be seen in these areas. The cells appeared swollen and granular. The blood vessels were filled with blood especially the capillaries of the glomeruli.

The HEART weighed 30.5 grams and appeared normal.

Microscopically, there was a diffuse connective tissue infiltration with mononuclear infiltration. There was an advanced cloudy swelling of the myocardial cells.

CASE 48—(5-5)

An unmarried woman, aged 45, was admitted with the complaint of "flooding spells." Profuse bleeding at each menstrual period and oedema after the period.

The FAMILY HISTORY was suggestive in that two brothers had died at 38 and 22 years respectively of "heart trouble."

In her PAST HISTORY she had had no rheumatic fever, chorea tonsillitis, scarlet fever, diphtheria, toxic goitre or syphilis.

Her PRESENT ILLNESS had begun 6 years before admission with swelling of the feet, legs, face, hands and abdomen, and dyspnoea without cough. One year later flooding spells had begun, occurring every 2 or 3 weeks, and lasting each time from 3 to 7 days.

The PHYSICAL EXAMINATION showed a well nourished woman. No cyanosis, dyspnoea or engorged neck veins were apparent. Marked carotid pulsation was evident. The lungs were negative. The heart was enlarged or displaced to the left and downward, extending 4 cm. to the left of the nipple line. The cardiac impulse was not seen but was felt in the sixth intercostal space 2 cm. to the left of the nipple line. No thrills were felt.

A harsh, grating systolic murmur was heard best over the 2d intercostal space near the sternum and heard also over the entire precordium. The pulse showed a quick rise and fall and was easily compressible. A capillary pulse was noted in the fingers. The arteries were somewhat thickened. The blood pressure was 112/50.

The spleen was not felt. The liver edge was at the costal margin. Pelvic examination revealed a submucous fibroid choking the vagina.

LABORATORY DATA: The urine contained a trace of albumin, but no casts. The blood showed a secondary anemia with hemoglobin 40 per cent; red blood cells 2,000,000;

white blood cells 5,000. The Wassermann was negative. The electrocardiogram showed the "Q.R.S." small in lead III, the rate 110 and the rhythm normal.

COURSE IN THE HOSPITAL: The patient was transfused for the secondary anemia and suffered only a slight reaction. The blood pressure remained low, 40/18. The patient developed an intercurrent bronchopneumonia and purulent pleurisy, and died the next day, that is, 19 days after admission.

The **CLINICAL DIAGNOSES** were: Bleeding submucous, myomata of the uterus. Severe simple anemia. Bronchopneumonia. Pleurisy.

The **PATHOLOGICAL DIAGNOSES** were: Multiple infected submucous, intramural and subserous myomata of the uterus. Purulent bronchitis. Diffuse bronchopneumonia. Left pleural empyema. Acute splenic tumor. Central necrosis of the liver. Cloudy swelling of the viscera. Slight hydropericardium. Cardiac hypertrophy. General arteriosclerosis. Epulis. An old amputation of the right leg below the knee.

THE GROSS AND MICROSCOPICAL DESCRIPTION.

The **KIDNEYS** were dark purplish red in color, looked enlarged and felt oedematous. The right kidney weighed 170 grams and the left 195 grams. Each measured 14x7x3 cm. The capsules stripped with ease. On section, the cortex as well as the medulla appeared congested.

Microscopically, the kidneys showed advanced cloudy swelling.

The **AORTA** was moderately sclerosed.

The **HEART** weighed 325 grams and appeared somewhat enlarged. Externally nothing abnormal was noted. The valves all appeared quite normal except for slight thickening in the aortic and mitral leaflets.

Microscopically, there was a moderate increase in connective tissue throughout the myocardium. In some parts there were areas of beginning calcification round which the cells showed advanced cloudy swelling. There were many small areas of simple necrosis. In many cells there was an abundant yellowish brown or granular hemofuscin pigment extending from the poles of the nuclei. The vessel walls were slightly increased in thickness.

CASE 49—(45-81)

An unmarried woman, aged 26, was admitted with the complaints of pain in abdomen, fever sweats and shortness of breath.

In her **PAST HISTORY** it was significant that at the age of 18 she had had rheumatic fever with which she was in bed one week and had stiff joints for a few months. She had had acute tonsillitis frequently.

The **PRESENT ILLNESS** had had its onset about twelve weeks before admission with headache, night sweats, afternoon fever, pain in the legs, weakness loss of strength and weight. She had had pain in abdomen for two months, first in the left side, then in the right side. The attacks of precordial pain were accompanied by palpitation and dyspnoea. All the symptoms, weakness and pallor had become progressively more marked.

The **PHYSICAL EXAMINATION** showed a patient looking quite sick. She was dyspnoeic and orthopnoeic, emaciated, pale and coughed frequently.

HEART: The cardiac impulse was in the 6th intercostal space. Auscultation revealed gallop rhythm, a snapping first sound and an apical blowing systolic murmur which was transmitted toward the axilla and also heard in the back. There was also a presystolic apical rumbling murmur and a loud, blowing, slightly rough diastolic murmur best heard in the third intercostal space on the left, transmitted from the aortic area to the apex. The pulmonary second sound was accentuated sufficiently to produce a diastolic shock. The cardiac dullness extended 4.5 cm. to right and 12.5 cm. to the left of the midsternal line. The heart dullness shifted very little on change of position. The rate was 132. The blood pressure was 95/45.

There was tenderness over the lower sternum and in the upper left quadrant and upper right quadrant. The liver was left 10 cm. below the costal margin. There were a few small brownish spots resembling purpura on the abdominal wall. The spleen was palpable to the level of the umbilicus.

The **LABORATORY DATA:** The urine was persistently negative except for a faint trace of albumin. The blood showed hemoglobin 55 per cent, red blood cells 3,440,000, white blood cells 10,800 on entrance, rose to 37,600. The Wassermann was negative. Electrocardiograms were normal. The pulse rate remained constantly around 130. The temperature was remittent between 99° and 104.6° with an afternoon rise. The temperature fell to normal during the last few days.

COURSE IN THE HOSPITAL: The patient failed rapidly. On the third day before death she developed dullness and suppressed breath sounds at both bases with crepitant rales at the left base and at the upper edge of the dullness. On the 10th day the patient complained of sudden precordial pain, the heart slowed, fluttered and stopped.

The **CLINICAL DIAGNOSES** were: Acute infectious endocarditis. Streptococcus viridans septicemia. Vegetations on the mitral and aortic valves. Chronic Myocarditis. Cardiac hypertrophy and dilatation. Mitral stenosis and insufficiency. Aortic insufficiency. Acute pericarditis with effusion. Infarcts of the spleen, lungs and kidneys. Chronic passive congestion of the liver. Bronchopneumonia. Bilateral pleural effusion.

The **PATHOLOGICAL DIAGNOSES** were: Chronic infectious streptococcus viridans endocarditis involving the mitral and aortic valves. Acute cardiac dilatation. General anasarca. Chronic passive congestion and cloudy swelling of the liver. Acute tubular nephritis. Acute splenic tumor. Infarction of the kidneys and spleen. Bilateral bronchopneumonia. Fibrous and calcified pulmonary tuberculosis. Meckel's diverticulum.

THE GROSS AND MICROSCOPICAL DESCRIPTION.

KIDNEYS: The right kidney weighed 140 grams and the left 150 grams. Each measured 13x5.5x2.5 cm. and the cortex measured 8 mm. The capsules stripped without difficulty exposing pale red, smooth surfaces and infarcts in the left. On section, grayish red

radiating lines could be seen in the pyramids and cortex. The glomeruli appeared congested.

Microscopically, the tubular epithelium was swollen and granular and occasional dilated tubules contained hyalin plugs. Small round areas of round cell infiltration were present in the cortex. Granular eosin staining material was seen within some of the glomerular capsules which were slightly distended. In some places a tubule was seen within a tubule.

The HEART appeared slightly enlarged and weighed 330 grams. A small, bright yellow elevated area about 2 mm. in diameter was seen in the epicardium over the left ventricle near the auriculoventricular junction. The valves measured as follows: Mitral 10 cm. aortic 6.5 cm., tricuspid 19 cm., and pulmonary 6 cm. The mitral valve was thickened with large irregular verrucous vegetations, which extended down along the chorda tendinae. The hydrostatic test showed the valve to be incompetent. The tricuspid valve was thickened and the hydrostatic test showed an insufficiency. The aortic valves presented large, irregular verrucous vegetations, the larger of which were about 8 mm. in diameter, especially on the anterior and posterior cusps. There were several minute perforating ulcers of the aortic valve cusps. The membranous septum below the septal cusp was weakened by a round depression. The defect, however, was closed on the right ventricular side by a thin wrinkled membrane, the septal cusp of the tricuspid valve. The aortic valve was incompetent to the hydrostatic test. The pulmonic flaps were smooth and competent.

MICROSCOPICALLY: Myocardium. There was a slight edema of the epicardium and of the stroma of the myocardium. The muscle fibers were often swollen and granular and appeared also to be fragmented in places. This tissue of the mitral valve was slightly oedematous and infiltrated by a few polynuclear leucocytes. Fresh and old vegetations were seen upon it. The former were made up of red blood cells, fibrin and polynuclear leucocytes. Numerous large colonies of cocci were seen in some vegetations.

There was a rather marked infiltration of polymorphonuclear leucocytes throughout the ventricular myocardium. There were areas of simple and coagulation necrosis and abscess formation. Advanced cloudy swelling and fatty infiltrative degeneration affected the myocardial cells which had escaped the other degenerative changes. There was an advanced fibroblastic infiltration. The subepicardial fat and connective tissue was increased and the seat of a very active inflammatory process. The myocardium along the epicardial border showed simple necrosis and fatty degenerative infiltration and colonies of cocci. The blood vessels showed congestion.

CASE 50—(12-12)

A bachelor, aged 64, was brought in on a stretcher with the complaint of pain in the abdomen.

His PRESENT ILLNESS had begun with abdominal pain two days before admission, becoming very severe in about six hours.

He had had paroxysms of acute abdominal pain at irregular intervals of 2 to 3 minutes, lasting for 15 to 45 seconds. The attacks were characterized by a drawing up of the knees close to the abdomen, with clonic convulsive movements, and a congestion or suffusion of the face from strain. The patient groaned and complained of severe abdominal pain. He had not had a bowel movement for two days. He had some nausea, but no vomiting.

The PAST HISTORY was negative except for a vague story of recurrent severe attacks of abdominal pain for four years.

The PHYSICAL EXAMINATION showed T. P. R. respectively 98.4, 84, and 26. Leucocytes 8,000. The urine was negative. The left pupil was twice as large as right, due to adhesions from trauma. The left pupil was irregular, eccentric and reacted poorly to light and accommodation.

Lungs were reported clear on admission. After the operation there were signs of a bilateral pneumonia.

The heart sounds were distant, no murmurs were heard. Reaction to the pain made the heart sounds indistinct. The blood pressure was 145/85. Brachial and radial arteries were thick and tortuous. The pulses were regular, equal, and synchronous with the apex beat.

The abdomen was distended, tympanitic and very tense during the attacks and moderately so between attacks. Peristalsis was visible in the upper abdomen moving downward. No masses or localized tender areas were made out.

The patient was given morphine 1/6 and atropin 1/150 and was operated under gas oxygen and ether anaesthesia. No intestinal obstruction was found. There was much gaseous distention of the bowel. There was a moderate amount of free fluid. The mesocolon was thick with oedema measuring about 1 cm. but decreasing during operation. No peritonitis was present. There was a small splenic hemorrhage. There were many congested areas. No volvulus was seen.

POST-OPERATIVE COURSE: About 18 hours after operation the pulse was noted to be irregular and weak. The patient was cyanotic and in collapse. A friction rub was heard in right axilla and base posteriorly. The temperature rose from normal to 102° at 12 noon. The bowels moved with an enema.

The chest began to fill up with rales which obscured the breath sounds. The abdomen was negative. The blood pressure was 90/60. The leucocyte count was 5,200. He was given 100 c.c. of 2 per cent glucose intravenously. The cyanosis increased. Respirations rose to 80. The pulse and heart rhythm became regular.

T. 104-2, P. 110, R. 20. The pulse became very irregular. Signs of pneumonia were noted at both bases posteriorly. Dr. F. N. Wilson reported pulse rapid, but regular with occasional extrasystoles.

The heart condition had apparently come on after return from the operating room.

The patient lost ground and died three days after admission.

THE CLINICAL DIAGNOSES WERE: Acute intestinal obstruction. Bilateral lobar

pneumonia. General arteriosclerosis. Chronic myocarditis. Transient auricular fibrillation. Cardiac decompensation. ?Syphilis. ?Angina abdominalis.

The PATHOLOGICAL DIAGNOSES were: Bilateral lobar pneumonia. Bilateral hydronephrosis. Dilatation of the bladder, both ureters and the urethra which was funnel shaped. Elongation of the sigmoid. Multiple hemorrhages into the peritoneum. Acute swelling of the spleen. Cloudy swelling of all viscera. Chronic fibrous tuberculosis, pleurisy and lymphadenitis. Chronic interstitial orchitis containing spirocheta pallida.

THE GROSS AND MICROSCOPICAL DESCRIPTION.

KIDNEYS: The right kidney weighed 140 grams and measured 11x6x2.5 cm., the left 175 grams and measured 12.5x6x3 cm. The cortex measured 5 and 6 mm. The capsules stripped with ease. The surface contained numerous large smooth sunken scars, but no adhesions. The stellate veins were congested. The gray columns were not definitely thickened. The pelves were dilated and filled with urine. Both ureters were dilated and the prostatic urethra showed a funnel shaped dilatation.

Microscopically, the convoluted tubules were slightly dilated. The lining cells were irregularly swollen and granular. The intima of a few of the larger arteries was thickened and hyalinized. The pelvic wall was thickened and contained a scattering of polymorphonuclear and small mononuclear cells. There were small areas deeper in the wall and the fat of the renal sinus which were densely infiltrated with these cells.

The AORTA showed numerous fatty plaques near its base. The coronary arteries were slightly tortuous, the walls were thickened with slight narrowing of the lumina.

The HEART weighed 320 grams. The epicardium contained considerable fat and showed a few small thickened areas in the epicardium of the left ventricle. Externally there was no evidence of myocardial change. The aortic valve flaps were uniformly thickened, but not shortened and appeared competent. There were two small fibrous nodules in the middle cusp. One was attached to the lower pole of the corpus aurantia. The other valves presented slightly thickened leaflets but were not otherwise abnormal.

Microscopically, there was some increase in the subepicardial fat and connective tissue. The blood vessels were dilated and showed marked acute congestion. There was a marked fatty atrophy in the subepicardial myocardium. In some of the myocardial cells there was advanced fatty degeneration infiltration, in others there was an abundance of hemofusin. Fibroblastic proliferation to a moderate extent was present throughout the myocardium.

CASE 51—(39-87)

A married man, aged 55, was sent in from the Out-Patient Department with the complaint of pain in the stomach for three months.

His FAMILY HISTORY and MARITAL HISTORY were irrelevant.

In his PAST HISTORY he had had measles followed by deafness in early childhood. He had had typhoid fever at 14, with jaundice. He had had a sore on the penis at 28, but no secondary lesions appeared. Gonorrhea had been contracted four times; the last time at 35. He had had pneumonia and pleurisy at 39 which kept him in bed for six weeks. He had had absolutely no gastric symptoms until the present illness began.

The PRESENT ILLNESS had begun four months before admission with a sudden onset of nausea about an hour after eating English walnuts. Soon after the vomiting, the first attack of "gnawing and grinding" epigastric pain began and ceased gradually within half an hour. Localized epigastric pain had been present every day since. Occasionally, when severe, the pain had radiated into the chest. It had been most severe two hours after meals and had been aggravated by vegetables, meat, and bread. He had relieved the pain with sodium bicarbonate and p-menthol or by vomiting. The pain had been almost constant for a month previous to admission. He had lost 22 pounds in three months.

The PHYSICAL EXAMINATION showed slight sallowness and emaciation. The tongue was heavily coated. There was pus about the teeth. The lungs were clear. The heart was 5.3 cm. to the right and 9 cm. to the left of the midsternal line. The aortic dullness measured 7.5 cm. The action was slow and regular. The P₂ was greater than the A₂; the M₂ was stronger than M₁. The A₂ was impure. A faint blowing systolic murmur was heard at the apex. The blood pressure was 140/82. The radials and brachials were fairly markedly thickened.

The abdomen was markedly rigid and tender in the epigastrium, especially toward the right. The K. K. was absent on the right and sluggish on the left. No A. K. was elicited.

The LABORATORY DATA: The urine was negative. The blood showed 4,840,000 red blood cells and 14,600 white blood cells per cu. mm., and 94 per cent hemoglobin. The Wassermann reaction was negative. The stools gave a positive benzidine reaction.

COURSE IN THE HOSPITAL: The patient suffered constant epigastric pain with more severe attacks, which were relieved by soda. Rigidity and tenderness persisted. He was transferred to Surgery where he had a sudden attack of very severe pain and was operated as an emergency. The perforation was closed and a gastroenterostomy was done. Four days later, the heart became very irregular and the patient became cyanotic, dyspnoeic, weak and irrational. A 15 c.c. dose of tincture of digitalis was given with good effects. The heart became regular twelve days after operation. Large furuncles developed and required incision and drainage. On the thirteenth day, dullness, bronchial breathing, crepitation and bubbling rales increased and bronchial voice sounds were noted over the right lower lobe. All stimulants failed. The temperature remained at 102° to 103°. The heart was regular but the rate rose to 120 per minute. The pulse became gradually weaker and the patient died nineteen days after admission.

The CLINICAL DIAGNOSES were: Perforated gastric ulcer, local peritonitis, gastroenterostomy, exhaustion psychosis, chronic myocarditis, transient auricular fibrillation, and bronchopneumonia of both upper lobes.

The PATHOLOGICAL DIAGNOSES were: Adenocarcinoma of the stomach, ulcerating and perforating, gastroenterostomy, infected abdominal wound with local peritonitis.

Bronchopneumonia with abscess formation, anthracosis. Chronic adhesive pericarditis, old pleural and peritoneal adhesions.

THE GROSS AND MICROSCOPICAL DESCRIPTION.

KIDNEYS: The right kidney weighed 230 grams and the left 240 grams. Each measured 13.5x6.5x3.5 cm. The cortex in each averaged 6 mm. in thickness. The capsules stripped with ease, leaving smooth surfaces which were of a deep red color. On section, the cortical striations were well defined. There was no streaking of the papilla.

Microscopically, there was a moderate congestion both of the vessels of the glomerular tufts and those outside the tufts. There was a small amount of granular material in the lumen of the tubule in many areas. The cells lining the tubules showed no change.

HEART: The pericardium was firmly adherent to the upper portion of the anterior surface of the right ventricle. There were about 35 c.c. of clear yellow fluid in the pericardial sac. With the parietal pericardium attached, the heart weighed 480 grams. The muscle was of good color and the valves had delicate margins free of thickenings. The aortic valve measured 9 cm., the pulmonary 8.5 cm., the mitral 10 cm., and the tricuspid 14 cm.

Microscopically, the epicardium over the right ventricle was conspicuously thickened and fibrosed. There was some moderately marked infiltration of connective tissue in the subepicardial myocardium. There was a slight general increase in fibroblastic elements throughout the muscle. Aside from moderate cloudy swelling, the myocardium showed no degenerative changes.

CASE 52—(60-66)

A boy, aged 16, was brought in because he had refusal to eat and could not sleep. The family and past histories were negative.

PRESENT ILLNESS: During the last year the patient had been acting queerly, but at no time had he refused to eat, and he had slept very poorly and had been restless at night. He had persecutory delusions about food and apparently many auditory hallucinations.

The **PHYSICAL EXAMINATION** showed a poorly nourished boy in whom no abnormalities could be made out.

The **LABORATORY DATA** were likewise quite negative. Blood: red cells 4,800,000, white cells 11,000, hemoglobin 85 per cent, differential count: 25 per cent lymphocytes and 70 per cent polymorphonuclears. The Wassermann was negative.

The urine was acid, with a specific gravity of 1.027 to 1.030. No sugar and only a faint trace of albumin and no casts were found. There were no acetone bodies present.

The spinal fluid contained no cells. The Pandy reaction was positive. The Wassermann was negative. The colloidal gold curve was normal.

COURSE IN THE HOSPITAL: On the eighth day, following a nasal feeding, the patient collapsed. He was cyanotic, with rapid, shallow respiration and a thready pulse. The entire abdomen was rigid. The lungs anteriorly were negative to auscultation. The percussion note was very tympanitic and drum-like over the left anterior, and a higher, shorter tympanitic note was heard over the left lower lobe posteriorly. Distinct bronchial breath sounds were heard in the lower lobe posteriorly and also in the right lower intrascapular area. There were also crepitant and snapping rales.

X-ray: Anterior-posterior view of the chest in the supine position was obscured by movement. There was a large amount of dense infiltration and extensive haziness along the descending bronchial branches on both sides, in appearance, suggestive of a pneumonia process.

The patient rallied somewhat during the afternoon, but became irrational about 8 P. M. Respiration gradually became more rapid and the pulse much weaker. The patient died nine days after admission.

The **CLINICAL DIAGNOSES** were: Catatonic dementia precox; aspiration bronchopneumonia; pneumothorax.

The **PATHOLOGICAL DIAGNOSES** were: Perforation of the oesophagus; left seropurulent pleurisy; bilateral bronchopneumonia.

THE GROSS AND MICROSCOPICALLY DESCRIPTIONS.

The **KIDNEYS** together weighed 250 grams and each measured 12x6.5x3.5 cm. The capsules stripped with ease, leaving a smooth surface. On section the cortex and medulla appeared normal. Microscopically: There was slight cloudy swelling of the tubular epithelium, but otherwise no abnormality.

The **AORTA** contained a few raised yellowish patches at the origins of the branches of the thoracic and abdominal aorta. Microscopically, a mild degree of atherosclerosis was noted, with slight thickening of the intima.

The **HEART** weighed 210 grams and appeared quite normal. The valves were normal and measured as follows: Aortic 6 cm., mitral 7 cm., pulmonary 6.5 cm., tricuspid 8.5 cm.

Microscopically, there was some simple necrosis and cloudy swelling. Congestion of both the arteries and veins was conspicuous.

CASE 53—(70-76)

An unmarried woman, aged 34, was admitted with the complaint of cough, pain in the chest, and difficulty in breathing.

The **PAST HISTORY** was of interest in that the patient had had pneumonia on three occasions, measles and whooping cough, and smallpox at 15.

The **PRESENT ILLNESS** consisted of a cough and cold for three weeks, which grad-

ually increased in severity. Three days before admission she had had a chill and a pain in the right side, and later in both sides. The following day her temperature was 103.8°. The breathing was rapid and difficult. A great deal of frothy sputum was expectorated.

The PHYSICAL EXAMINATION revealed the patient propped up in bed, with distinct cyanosis of the lips and finger tips, and a pinched expression. She was coughing frequently and bringing up frothy mucopurulent sputum, but no blood.

Lungs: The percussion note was dull on both sides posteriorly, below the level of the 6th spine and on the right anteriorly below the 5th rib. Coarse bubbles were heard over both lower lobes posteriorly. Many coarse rales were present over the entire chest anteriorly. A friction rub was noted at the right base. The breath sounds were bronchovesicular anteriorly and bronchial at the right base posteriorly. Some fine crepitant rales were heard here also.

The heart sounds were moderately strong and the rhythm was regular and the P2 was not accentuated. There were no murmurs or adventitious sounds. The radial pulse was of medium size. The blood pressure was 130/70.

LABORATORY FINDINGS: The urine contained a large amount of albumin (0.3 g. to 1.4 g. per liter), and many fine granular and some fine hyaline casts, later also some red blood cells. The chloride content was 1.1 gm. per liter.

Blood: R. B. C. 4,740,000; W. B. C. 10,600 on admission to 46,000 at death; hemoglobin 80 per cent. The differential count was within normal limits.

The tuberculosis complement fixation test was negative. The Wassermann was negative. Blood cultures were negative. Sputum cultures showed B. Influenza predominating. The sputum contained no tubercle bacilli.

The x-ray plate was diagnosed pulmonary tuberculosis of the miliary type. The cardiac shadows extended 4 cm. to the right and 8.5 cm. to the left of the midsternal line. The aortic shadow measured 5 cm.

COURSE IN THE HOSPITAL. The patient seemed better at times, but gradually grew worse. The temperature, pulse, and respiration fluctuated somewhat, T. 101°-104°; P. 110-140; R. 40-60. She died nine days after admission.

The CLINICAL DIAGNOSES were: Bilateral influenzal bronchopneumonia; edema of the lungs; lung abscess; acute nephritis.

The PATHOLOGICAL DIAGNOSES were: Bilateral influenzal bronchopneumonia with peribronchial consolidation; purulent bronchitis; fibrinous pleurisy; fibrinous perisplenitis and perihepatitis; focal necrosis of the liver; cloudy swelling of the kidneys caseous tuberculous nodules of the right peribronchial lymph glands; calcified tubercles of the spleen and liver; Meckel's diverticulum; lung and bronchial cultures contained B. Influenzae.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS.

The KIDNEYS together weighed 440 grams and each measured 14x7x4 cm. The capsules were slightly adherent, small bits of kidney tissue clinging to the stripped capsule. An occasional renal cyst was noted. The kidneys appeared swollen.

Microscopically, there was cloudy swelling. The renal epithelial cells appeared granular. Occasional hyaline casts or granular sediment were found in the tubules. Two small renal cysts were present. There was some edema of the interstitial tissue. Small infected thrombi filling the lumina only incompletely, were present in a number of veins.

The HEART weighed 205 grams and externally there were no evident abnormalities. The valve leaflets were thin and normal and the valve rings were not stretched.

Microscopically, there was advanced cloudy swelling of the myocardial cells with simple necrosis in some areas. Some of the blood vessels were gorged with polymorphonuclear leucocytes, some of which had penetrated the walls and were scattered in the myocardium and had accumulated in certain areas.

CASE 54—(20-165)

A widow, aged 42, was admitted with the complaint of tumor masses in the epigastrium and in the left groin. She had also had pain in the masses, shortness of breath, and dizziness.

The FAMILY HISTORY was of interest in that her father and two brothers had died of Bright's disease.

The PAST HISTORY was irrelevant. She had had frequent attacks of tonsillitis, pneumonia at 11, and a fractured right tibia and fibula at 36.

The PRESENT ILLNESS had begun two years before admission with nervousness and insomnia. She had undergone severe prolonged nervous strain following the illness and death of her husband from angina pectoris. Bromides had given her some relief. She had also noted a loss of strength and weight. She had had attacks of vertigo for one and a half years. A mass had been felt beneath the left rib margin one year previous to admission. The mass had gradually increased in size and had become tender and painful. Shortness of breath and edema of the feet and ankles had been present for some months. She had lost fifty-three pounds in weight in two years.

The PHYSICAL EXAMINATION showed a pale, but still fairly well nourished woman. The teeth were very poor and four alveolar abscesses were found.

The neck and chest were negative. The lungs were clear and resonant.

Heart: The cardiac impulse was not seen or felt. No thrills or shocks were noted. The dullness extended 2.5 cm. to the right and 11 cm. to the left in the 5th interspace. A systolic murmur was heard at the apex. The heart sounds were clear.

The abdomen contained a moderately hard mass in the left upper quadrant. The mass was smooth with a deep notch on the medial side. It moved down with inspiration. A small mass of glands was present in the left groin.

The LABORATORY EXAMINATIONS showed: Blood: hemoglobin 66 per cent. red blood cells 3,300,000, white blood cells 320,000. The differential count showed 26 per

cent myelocytes, 62 per cent polymorphonuclear neutrophils, 8.5 per cent endotheliocytes, and 8.5 per cent lymphocytes. The Wasserman reaction was negative. The blood sugar was 0.135 gm. per 100 c.c. The non-protein nitrogen was 75 mg. and the urea 8 mg. per 100 c.c. of blood.

URINE: No sugar, albumin, casts, or cells. The 'phthalein excretion amounted to 58 per cent in two hours.

Films of the teeth showed an abscess at the roots of the upper right lateral cuspid.

The electrocardiograms showed left ventricular preponderance.

COURSE IN THE HOSPITAL: The white blood cell count dropped to 21,000, with only 3 per cent myelocytes. The spleen could not be felt with the patient lying down, but the edge was just palpable with the patient on the right side.

About 18 months after the onset of her trouble she was admitted for the fourth time. She had had constant pain in the epigastrium, which was worse after eating. A mass had been felt in this region. The pain in the left groin was comparable to "labor pains" and increased on exertion.

A general glandular enlargement had begun three months before admission, with the glands in the neck the first to be involved. These subsided with x-ray treatment.

Profuse menstruation, three weeks at a time, had been troublesome since the onset of the glandular enlargement. All the glands had become enlarged within two months. She had gained to 190, but had lost ten to twelve pounds of weight recently.

There was a mass in the epigastrium, tender and hard and attached to the posterior wall extending from the umbilicus out to the nipple line and the left parasternal line up to the level of the costal margin. The glands in the inguinal region were enlarged and these were larger on the left where the patient felt the pain. There were glands of all sizes in the neck, discrete, hard, and smooth, and loosely attached. There were glands in both axillae. The glands in the inguinal region were the largest, some measuring $1\frac{1}{2}$ cm. in length.

The spleen was not felt. The liver dullness extended just to the costal margin.

Masses were found on both sides of the pelvis closely attached to the pelvic wall, beginning along the sides of the ileum and seemingly lost in the pelvis.

A section of the lymph gland excised for diagnosis was reported to show a leukemic process. The spleen became palpable at the end of inspiration and ten days later extended 4 cm. below the costal margin, and later to the umbilicus. It was hard and thick.

The patient suffered considerable pain, which was colicky in character. The constipation was obstinate and distention was marked. It was relieved to some extent by asafetida enemas. At times the patient would have attacks of vertigo and sinking sensation in which she thought she was going to die. There was profuse perspiration at times. The patient's erythrocytes and hemoglobin steadily came down. Her condition gradually became worse. She became progressively weaker and the masses in the neck, axilla, and abdomen became progressively larger. Respiration became shallow and frequent and the pulse became weak and the patient died two months after her last admission and 18 months after the onset of her trouble.

The CLINICAL DIAGNOSES were: Aleukemic leukemia, severe secondary anemia, tympanites, ascites, mitral regurgitation, subcutaneous hemorrhages.

The PATHOLOGICAL DIAGNOSES were: Multiple lymphomata involving the lymph glands and liver, enlargement of the spleen, central necrosis of the liver lobules, ascites, hydrothorax, hydropericardium, edema of the lung with congestion, cloudy swelling of the viscera, bilateral fibrous adhesive pleurisy and peritonitis, chronic cholecystitis, cholelithiasis, cystitis, cervicitis and vaginitis.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS.

The KIDNEYS weighed 150 grams each and measured $12 \times 7 \times 3.5$ cm. The capsule of the left kidney was adherent. On section the pelvis of each was filled with purulent urine and the columns were congested.

Microscopically, the cells of the tubules and glomeruli were swollen and granular. The nuclei stained poorly or were absent. There were areas of scarring and lymphoid and polymorphonuclear infiltration.

The AORTA contained numerous fatty plaques around the openings of the vessels.

The HEART was large and flabby and weighed 400 grams. No incompetencies were made out by the hydrostatic test. There were no vegetations and no evident valvulitis. The valves measured as follows: Mitral 9 cm., aortic 7 cm., tricuspid 10 cm., and pulmonary 6.5 cm. The tiger lily, or thrush-breast barring was evident on the papillary muscles.

Microscopically, there was an advanced simple necrosis and cloudy swelling with a diffuse mononuclear infiltration. Some areas showed a fatty degenerative infiltration. The arterioles were sclerotic. The epicardial fat was excessive. The endocardium was thickened and distinctly fibrosed in places. A few leukemic clots were noted in the sections.

CASE 55—(2-2)

A Belgian, aged 49, was brought in in an ambulance complaining of inability to breathe at night and difficulty in breathing at all times; also pain in the chest and poor vision to the left eye.

The FAMILY HISTORY contained no significant facts.

The PAST HISTORY was significant. He had had measles and whooping cough in infancy. He had contracted syphilis at 23 as a "hard chancre" with no secondary lesions. At 23 he was operated for a "bubo" (blue ball). He had also had an operation for hemorrhoids in 1916. Otherwise, he had been well up to the present time.

MARITAL HISTORY: He was married at 28 (in 1898). His wife had had no miscarriages. Three children were dead; one died at three months, of pneumonia, one at 11

months and one at 8 months of unknown causes. His wife and five children were living and well.

The patient had done hard manual labor. He had been a hod-carrier until 7 months before admission.

The PRESENT ILLNESS was dated from the failure in eye-sight which had begun insidiously about 4 years previously and had progressed slowly. Pain in the chest had developed, radiating from the right edge of the sternum to the anterior axillary line and posteriorly to the interscapular region. It had been present for about 7 months. Dyspnea had gradually increased in severity. Hoarseness had been present for about 6 months. A cough with occasional expectoration had caused an increased dyspnea. For three or four nights before admission he had been unable to recline, but had to sit up constantly and had been extremely dyspnoeic. His appetite had been good up until the present illness had begun to be severe. He had lost 40 pounds in weight in two years and had been very weak and unable to work for 7 months.

The PHYSICAL EXAMINATION showed him sitting up in bed. His breathing was very labored. He was actually gasping for breath and could not lie down. There was audible wheezing, pallor, and slight cyanosis of the lips. He talked only between breaths and suffered paroxysms of slightly brassy unproductive coughing. The pupils were slightly irregular but reacted to light and accommodation. The mucus membranes were pale. The teeth were very poor. There was extreme pyorrhea and the gums were slightly red. In the neck, the jugular veins were engorged and there was marked conspicuous carotid pulsation. There was a suggestion of tracheal tugging. The chest was barrel-shaped. The left upper anterior chest bulged. The percussion note was impaired over the bulging area, dullness extending to the 6th dorsal level. There was slight lagging of the left upper chest, otherwise expansion was good. No heave was felt, there was, however, a diastolic shock over the bulge. Pulsation was noted in the left axilla and epigastrium. Inspiration and expiration were difficult and wheezing was heard anteriorly and posteriorly over most of the chest. The breath sounds were much suppressed over the left upper anteriorly, and harsh posteriorly. Sibilant and sonorous rales were present.

HEART: A slight heave to the precordium was evident. The cardiac impulse was not seen. The aortic dullness measured 10.5 to 14 cm. The dullness extended 4 cm. to the right and 9 cm. to the left in the 5th interspace. Slight irregularity of the rhythm was noted at times. The radial was slightly irregular and small.

There was a distinct disproportion between the volumes of the radial pulses on the two sides. The blood pressure 122/90 on the right and 108/90 on the left side.

The abdomen was negative.

The reflexes were all sluggish. The fingers were slightly clubbed. The nailbeds were pale and slightly cyanotic.

LABORATORY DATA: The urine was not at all abnormal. The phthalein excretion was 50 per cent the first hour and 10 per cent the second hour.

The blood examination showed 90 per cent hemoglobin, 5,200,000 red blood cells, and 7,400 white blood cells. The differential count was normal. The Wassermann was four plus.

The electrocardiograms showed a questionable right ventricular preponderance. The complexes were small in all leads. The P waves in lead III were negative.

COURSE IN THE HOSPITAL: The patient gradually lost ground, and choking attacks became more severe, and finally caused exitus twenty days after admission.

The CLINICAL DIAGNOSES were: Aneurysm of the aorta. Syphilis. Chronic myocarditis. Chronic bronchitis. Paralysis of the left vocal cord. Chronic laryngitis. Optic atrophy.

The PATHOLOGICAL DIAGNOSES were: Syphilitic aortitis with aneurysm of the arch and descending aorta. Pressure closure of the left bronchus and trachea. Scar of an old gumma of the liver. Periportal infiltration. Chronic orchitis. Chronic fibrous perisplenitis. Numerous calcified nodules in the spleen, in both lungs and in the peribronchial glands. Hyperplasia of the splenic corpuscles. Acute congestion of the liver.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS.

The KIDNEYS were of about normal weight and size. The capsules were adherent in a few areas and the surface was torn in these places. The surface was otherwise pale grayish white, except for the areas of congested stellate veins. The kidney on section appeared slightly oedematous and the markings were obscured.

Microscopically, the kidneys presented the picture of an advanced parenchymatous degeneration of the epithelium of the convoluted tubules, and a marked congestion of the arterioles. There was some perivascular round cell infiltration and a few small scars.

The AORTA was the seat of much pathology and the ascending aorta was greatly dilated. There was a sacular aneurysm of the descending arch of the aorta, which was filled with a firm but friable clot. The aneurysm extended down to the level of the fourth rib and to the right margin of the sternum and filled that portion of the thorax included in these boundaries. It appeared to rise from the posterior wall of the descending part of the arch.

The HEART was small and flabby. There was nothing abnormal externally, and the valve leaflets were slightly thickened, but otherwise not abnormal.

Microscopically, there was considerable patchy fibrosis, endarteritis and some perivascular mononuclear infiltration fibroblasts and angioblastic proliferation, brown atrophy, and a chronic passive congestion. There was some infiltration of the epicardial fat into the myocardium. The endocardium was somewhat thickened in some sections.

CASE 56—(43-87)

A man, aged 61 years, was admitted with the complaint of headaches of three weeks duration, associated with weakness, loss of weight, and difficulty in urination.

The FAMILY HISTORY and the PAST HISTORY contained no significant statements. No suggestive symptoms had been elicited.

The PRESENT ILLNESS had apparently begun three weeks before admission with headache, which was limited to the right side of the vertex. The eyes had felt sore for a few weeks. There had been some loss of appetite and some nausea. Weakness had compelled him to quit his work two weeks before admission. Nocturia, difficulty of, and burning on urination, which had been troublesome for some years, had become exaggerated.

The PHYSICAL EXAMINATION showed a pale, somewhat emaciated old man of fairly large stature. Sallowiness and drowsiness added to the picture that suggested chronic sepsis.

There was some weakness of the left facial nerve. The temporal arteries were sclerosed and tortuous. The oral hygiene was very poor.

The heart was not enlarged, a systolic murmur was heard at the apex. The blood pressure was 108/70. The lungs contained some moisture at the apices, the rales however were variable.

The abdomen was held too spastic for accurate palpation. The prostate was slightly enlarged and tender.

LABORATORY EXAMINATIONS showed a secondary anemia and a leucocytosis (Hb. 60 per cent, R. B. C. 3,300,000; W. B. C. 26,000). A differential count showed 90 per cent polymorphonuclears. Blood cultures were repeatedly negative. The Wassermann reaction was negative.

The urine contained a trace of albumin, a few hyalin casts and a few leucocytes. The phthalein excretion was normal. The blood non-protein nitrogen was 32.3 mg. per 100 c.c.

The electrocardiograms, X-ray plates of the skull and chest and the perimeter fields were normal.

COURSE IN THE HOSPITAL: The pulmonary symptoms increased slightly while the patient was in the hospital. Nine teeth were extracted.

The temperature was normal in the morning, but up to 100° in the afternoon and later often reached 102.2°.

Early in the morning of his 28th day in the hospital the patient developed a sudden pain in the epigastrium with nausea, but no vomiting, with, however, increased rigidity in the upper abdomen, especially on the right. Nothing was palpable. The patient was tender to a quick jab over the gall bladder region. There was no jaundice. The temperature was 100° and the leucocytes were 22,400. Laparotomy was advised but the consent was not obtained until 7:30 P.M., ten hours later.

At operation the abdomen contained a small amount of clear fluid with flecks of fibrin. The liver was large and edematous. The finger entered the foramen of Winslow easily. and no pus was found in the lesser peritoneal cavity. The stomach was apparently normal. The gall bladder was thickened and adherent to the duodenum, but not distended and contained no stones. A few enlarged soft glands were felt along the common duct. The appendix was thickened and edematous. The tip was densely adherent in a semicircle to the caecum. The process was evidently of 5 or 6 weeks duration. The appendix was removed and the stump buried. Tube drainage of the gall bladder was instituted. A cigarette drain was inserted under the gall bladder. The abdomen was closed. Pathological examination showed acute inflammation in the appendix.

POST-OPERATIVE COURSE: The leucocytes gradually dropped from 26,000 to 16,000 and as the patient became moribund to 12,000 and 8,750. The patient was non-communicative, dull, but the condition was apparently good. The drainage of bile averaged 100 to 150 c.c.

There was pain on coughing. The percussion note was dull and rales were heard over the apices. Chills with febrile rises to 104° were occasionally present.

The complexion became sallow. A trace of bile was present in the urine and the feces were light yellow. There was no definite jaundice, but the drainage of bile lessened materially. The patient voided 50 to 150 c. c. of urine every hour. The patient continued growing progressively weaker. He vomited after practically every feeding, thus he retained very little nourishment.

The wound remained clean. Bile was draining freely from the abdominal wound. The lung signs were not important. The abdomen was always soft. The temperature ranged between 97° and 100°. The pulse was rapid, averaging around 120, and became weak. He died two months after admission.

The CLINICAL DIAGNOSES were: Cephalalgia. Acute appendicitis. Chronic cholecystitis with an acute exacerbation. Perihepatitis. Hepatic pyelophlebitis. Enlargement of the liver. Chronic bronchitis. Severe secondary anemia. Arteriosclerosis.

The PATHOLOGICAL DIAGNOSES were: Perforated gastric ulcers of the greater and also of the lesser curvature, with localized peritonitis. Subdiaphragmatic abscess on the left penetrating into the liver. Multiple liver abscesses. Drainage of the gall bladder and appendectomy. Acute and chronic cholecystitis. Fibrinopurulent perisplenitis. Purulent bronchitis. Thrombosed pulmonary vein in the left lower lobe. Septic pneumonia in the right and left lower lobes. Interlobar adhesions on the right. Acute tubular nephritis. General arteriosclerosis. Calcification of the aortic valve. Caseous and calcified peribronchial tubercular lymphadenitis.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS.

The KIDNEYS were congested and firm with gray white opaque areas. The cortical striations were lost in many places. The posterior half of the right kidney was entirely gray white and opaque in appearance. There were several retention cysts. The kidneys weighed 520 grams and each measured 12x8x5 cm.

Microscopically, there was degeneration of the tubular epithelium with swelling, granulation and granular debris within the lumina and Bowman's capsules. There were numerous hyaline and epithelial cells casts. In some tubules the pitheal cell casts contained lumina within the cast. There was an abundance of fat, especially in the convoluted

tubules. There was oedema of the interstitial tissue with an infiltration of lymphocytes in places.

The AORTA showed generalized sclerosis. The abdominal part especially contained numerous plaques with ulcerations. The pulmonary artery showed some mild sclerotic changes.

The HEART: The pericardial fluid was slightly turbid and slightly increased in amount. The heart weighed 200 grams. The mitral valve showed slight regurgitation by the hydrostatic test. The mitral and aortic valve leaflets were slightly thickened and sclerotic. The valve rings measured as follows: Mitral 10.5 cm., aortic 7.5 cm., tricuspid 13 cm. and pulmonary 7 cm.

Microscopically, there was a slight diffuse fibrous tissue increase, with sclerosis and tortuous blood vessels. There was slight mononuclear and polymorphonuclear leucocytic infiltration, especially marked in the subendocardium with thickening and hyalinization of the endocardium and subendocardium.

CASE 57—(56-70)

A truck driver, aged 43 years, was admitted with the complaints of loss of appetite and weight, and shortness of breath.

His PAST HISTORY was not very significant. He had had an acute appendix removed thirteen years before admission. He had suffered an attack of "grippe" five years before and a mild attack of bronchitis one year previous to admission. He had had a chronic nasal catarrh for years.

He had been a copper and gold miner for 14 years and had for a few years been a truck driver. He had always worked hard and had been exposed to wet and rainy weather many times during the past four years. He had noticed increasing weakness for a few months.

PRESENT ILLNESS: Three weeks before admission, after a hard days work, he went to bed aching all over and had a chilly feeling. The next morning he was too weak to walk and consequently remained in bed. He had fever, from the onset, ranging as high as 101°, two weeks before admission. Since then it had been irregular but always highest in the evening. He had had severe drenching sweats for two or three nights. Gradually he became weaker, his appetite failed and his bowels became constipated. Only occasional cough was noticed and very little sputum was produced. He had lost some weight, felt tired all the time, suffered from afternoon fever with sweats, shortness of breath, loss of appetite, and nocturia.

The PHYSICAL EXAMINATION showed a cyanotic flush of the cheeks and neck. The skin was hot and moist. The temperature was 102.5°. The patient was fairly well nourished. The neck showed slight pulsation on either side. The thorax was hyposthenic. The chest expansion was good. There was no Litten's sign. The percussion note was impaired at the right apex. The breath sounds were harsh and increased over the apices with prolonged expiration over the same regions. No rales were heard. The whispered and spoken sounds were increased over the right side posteriorly down to the level of the 8th spine.

The heart was not enlarged. The dulness extended 3 cm. to the right and 11 cm. to the left of the midsternal line. The heart sounds were of good quality, clear and regular at a rate of 100 per minute. The pulmonary second sound was louder than the aortic second sound. No murmurs were heard. The blood pressure was 125/80.

The abdomen and extremities were negative.

Ophthalmoscopic examination revealed a yellowish-white spot measuring one-third of the disc diameter, along the superior temporal vein in the left fundus.

LABORATORY FINDINGS: Blood: Red blood cells 4,600,000. White blood cells 4,550. Hemoglobin 75 per cent. Differential: 29.5 per cent Lymphocytes; 10 per cent large Mononuclears; 60.5 per cent Polymorphonuclears. The Wassermann and Tuberculous complement fixation tests were negative. The blood cultures were negative on eight occasions.

URINE: Acid. The specific gravity was 1920. No sugar. A trace of albumin was present at times after the ninth day. No casts or red blood cells were found in the sediment. The day and night quantities were about equal, with a variation of 10 points in the specific gravity. The "phthalein" excretion was 40 per cent the first hour and 25 per cent the second hour. Urine cultures were negative. The stool examination and culture were negative. The sputum was very small in amount and mucopurulent in character. No elastic tissue or tubercle bacilli could ever be found. The sputum cultures showed a large variety of organisms.

Electrocardiograms revealed a suggestion of a right ventricular preponderance, with large P waves.

X-ray of the chest showed pleural thickenings at the costophrenic. The lung fields were almost totally opaque. The greatest density was toward the axilla. The remainder of the lung showed a rather coarse parenchymatous mottling. X-ray diagnoses were: Pulmonary tuberculosis, both pneumonic and miliary.

COURSE IN THE HOSPITAL: On admission the temperature was 102°; pulse was 80 and respiration 22. White blood cells 4,550. The temperature continued high but intermittently down to 100° and 101° in the morning and up to 102.5° and 103° in the evening. The signs in the chest gradually increased. The percussion note became impaired over the upper part of the right lung and the left apex. Fine rales were heard in both paravertebral spaces, and whistling soft ronchi. The spleen was palpable after eight days but could be felt for only two days. Seven days after admission the patient was irrational at times, most markedly so at the height of the fever.

Respirations gradually became more rapid and difficult. The lips and nail beds became

more cyanotic. The signs in the lungs continued to increase, more rales were heard with superficial crackles, inspiratory and expiratory in type. The breath sounds were harsh. The leucocytic count remained low, 5,800, 11,800, 7,200.

The mental condition gradually became worse with the increase in the signs and the patient became very restless, irrational, delirious at times. No symptoms of meningeal involvement appeared.

The pulse gradually became weaker and more rapid, and the respiration very difficult. The cyanosis increased. He died ten days after admission.

The CLINICAL DIAGNOSES were: Tuberculous bronchopneumonia, tuberculous chondritis, miliary tuberculosis, fibrinous pleurisy, chronic passive congestion of the kidney. Dental caries.

The PATHOLOGICAL DIAGNOSES were: Anthracosis of the lungs and peribronchial lymph nodes (coal miners' lung) with carbon deposits in the liver, spleen, pancreas and adrenals. Acute disseminated tuberculosis of the lungs with consolidation. Miliary tuberculosis of the pericardium, liver, spleen, kidneys, pancreas and adrenals. Chronic passive congestion of the liver, spleen, kidneys and intestines. Hypertrophy of the adrenal cortex. Chronic fibrous pleuritis, chronic emphysema. Small fibrous adhesions of pericardium to anterior to anterior thoracic wall.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS.

The KIDNEYS weighed 450 grams. On section there were round or irregularly shaped white areas in both cortex and medulla. The mucosa of the pelves appeared normal. Microscopically, the spaces within Bowman's capsule were somewhat dilated and contained a small amount of granular material. The lining cells of the convoluted tubules were swollen, granular and had ragged edges. Their lumina contained cellular debris. In the cortex were several areas in which the glomeruli and tubules were replaced by a pink staining homogenous material surrounding which were lymphocytes, fibrin and epithelial cells.

The AORTA showed slight atheromatous degeneration about the openings of the coronary arteries.

The HEART weighed 360 grams. The epicardium showed miliary tubercles along the branches of the coronary vessels. The valve leaflets were all thin and delicate and competent by the hydrostatic test. The myocardium was flabby and light in color. Microscopically, the epicardium contained occasional tubercles. The myocardial cells were granular and swollen. The blood vessels walls were thickened and hyalinized. There was a diffuse mononuclear leucocytic infiltration with occasional groups of these cells. A slight subendocardial fibrosis was present.

CASE 58—(23-106)

A boy, aged 21, was admitted complaining of shortness of breath, hoarseness and swelling of the legs.

His FAMILY HISTORY was of interest in that his father and brother had had heart disease.

In his PAST HISTORY he had had pneumonia at three years and measles at eight years. He had had acute rheumatic fever at twelve years with red swollen and painful feet and ankle joints for two years. He had not had fever or cardiac complications recognized as such at that time. He had had a second attack at fifteen years.

At the age of nineteen he had had inflamed joints and fever which had confined him to bed for one month. He had been told during this attack that his heart had been involved.

His PRESENT ILLNESS had been dated from the onset of shortness of breath and palpitation, which had been noticed eight or nine months before admission. Five months before admission he had had "pneumonia," during which disease his fever rose to 105°. He had been very sick for about three weeks, but he had never recovered entirely. The shortness of breath had been exaggerated and in addition he had developed precordial pain, hoarseness, a productive cough, and oedema of the legs.

The PHYSICAL EXAMINATION showed a tall boy propped up in bed, slightly dyspnoeic and slightly cyanotic. He coughed after any exertion.

Th tonsils were small and submerged but contained fluid pus. The epiglottis and the arytenoids and the vocal cords were congested. The movement of the right vocal cord was sluggish.

The neck veins were only slightly engorged.

The lungs were negative. The left chest bulged anteriorly.

HEART: There was a slight systolic heave of the bulging precordium. The cardiac impulse was felt in the sixth interspace 15 cm. to the left of the midline. A systolic retraction of slight degree was seen in the fifth interspace. A well defined diastolic and systolic thrill was felt over the impulse. A diastolic shock was palpable in the pulmonary area. The cardiac dullness extended 5 cm. to the right and 12 cm. to the left of the midline. The aortic dullness measured 6 cm. in width. A soft high pitched aortic diastolic murmur was heard along left sternal border. A long rumbling apical diastolic murmur preceded the first sound and a high pitched systolic murmur followed the sharp first sound at the apex. The pulmonary second sound was strongly accentuated, while the aortic second sound was very faint and distant. The rhythm was absolutely irregular and a slight pulsus deficit was present. The blood pressure was 120 to 140 systolic and 50 to 60 diastolic.

The abdomen was distended and tense. The liver extended 8 cm. below the costal margin in the midclavicular line and the tenderness to pressure was very easily elicited. The spleen was not felt. No signs of fluid in the peritoneum were found to be present.

The extremities were negative. No pistol shot sound, Duroziez sign, capillary or Corrigan pulse was demonstrated. The reflexes were normal.

The LABORATORY EXAMINATION showed: The urine contained .5 gram of albumin per liter and many hyaline and granular casts. The twelve hour volumes were low. The phthalein excretion amounted to 75 per cent in two hours.

The blood examination showed 75 per cent hemoglobin, 4,400,00 red blood cells, and 8,500 white blood cells. The differential count revealed nothing abnormal except a slight increase in eosinophiles totaling 2 per cent. The Wassermann reaction and repeated blood cultures were negative.

The electrocardiograms showed right ventricular preponderance and auricular fibrillation with coarse waves.

COURSE IN THE HOSPITAL: The patient was kept digitalized, but nevertheless he had frequent attacks of precordial pain and also hemoptysis, anorexia, nausea, and epigastric pain. The engorgement of the liver persisted.

The tonsils were removed, but the patient reacted very poorly to the operation. He complained of severe pain in the abdomen, nausea and vomiting. The irregular heart action was exaggerated, the rate rose to 160 per minute and the pulsus deficit increased to 60. A troublesome hiccough developed. He became drowsy and irrational and died on the third day after the operation, which was two months after admission.

The CLINICAL DIAGNOSES were: Chronic cardiac valvular disease, mitral stenosis and insufficiency and aortic insufficiency. Auricular fibrillation. Chronic myocarditis. Chronic interstitial nephritis. Chronic passive congestion of the viscera. Pulmonary infarcts. Heart failure with congestion and oedema.

The PATHOLOGICAL DIAGNOSES were: Chronic mitral, aortic and tricuspid endocarditis with acute vegetations. Mitral stenosis. Cardiac hypertrophy and dilatation. Chronic passive congestion of the viscera. Chronic fibrous pericarditis and bilateral obliterative pleurisy. Caseous tuberculosis in the left apex and the peribronchial lymph nodes. Blood cultures and cultures of the vegetations were sterile.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS.

The KIDNEYS were large and horse shoe shaped, the right measured 13.5 cm. and the left 11 cm. The organs were very firm and cut with some difficulty. The capsules were not adherent but appeared slightly thickened. On section blood dripped freely from the cut surface. The striations were very indistinct. The glomeruli were prominent and deep red in color. The tubular portions were opaque and gray.

Microscopically, the capillaries of the glomeruli were distended with blood. There was a sediment in Bowman's capsule and in many of the tubules. The tubules showed a moderate degree of cloudy swelling in that epithelial cells were granular and cloudy.

The AORTA was somewhat inelastic and thickened at its root. The coronary arteries, however, were negative. Microscopically the aorta showed thickening of the intima with considerable vacuolization of the media, the architecture of which was practically destroyed by this vacuolization. The pulmonary artery showed intimal thickening.

HEART: The pericardium was adherent to the chest wall and the lung. The pericardial cavity contained 70 c.c. of clear straw colored fluid.

The heart was very large and consequently occupied a large part of the left chest. All the cavities of the heart, especially the left auricle and to an almost equal extent the right auricle and the great veins and even the ventricles were engorged with blood and greatly dilated. The heart was also definitely hypertrophied, the muscle weighing 480 grams.

The wall of the dilated left auricle showed evidence of hypertrophy and measured 3 to 4 mm. Small firm nodular thickenings of the endocardium were visible. In the left auricular appendage there were some gray mural thrombi adherent to the muscular trabeculae. The right auricular wall showed some hypertrophy, measuring 3 mm. in spite of the dilatation.

The mitral valve was thickened and firmly contracted, leaving only a narrow slit like opening. The two cusps were fused together at the angles. At the left angle there were a number of vegetations many of which were calcified. A few, the largest of which measured 3 mm. in diameter, were softer and apparently fresh. The vegetations had a warty appearance, most of them having broad bases. There were a number of minute vegetations about the entire ring. The tricuspid valve measured 13.5 cm. and in position admitted four fingers. There were a few small, the largest measuring 2 mm. in diameter, smooth, warty vegetations along the free margins. The aortic valve ring measured 6 cm. The valve leaflets were thickened and contracted producing a definite insufficiency. There were a few small hard vegetations along the valve borders. The pulmonary valve measured 6 cm. and the leaflets appeared slightly thickened but otherwise normal.

Microscopically, the auricular muscle was definitely hypertrophic and showed fragmentation. The endocardium was very much thickened due to layers of homogenous pink staining tissue and fibrous tissue. The tricuspid valve leaflets showed small well organized vegetations. There was thickening due to many layers of fibrous tissue which contained only a few connective tissue nuclei. The aortic valve leaflets were definitely thickened and showed lymphocytic accumulations in the intima and adventitia, but not about the blood vessels.

The ventricular epicardium was slightly thickened. The myocardium was greatly hypertrophied, and showed much fragmentation and fibrosis. Besides diffuse fibrosis there were many patches with mononuclear infiltration. Some areas suggested Aschoff nodules. There was an advanced cloudy swelling with simple necrosis and fatty infiltration degeneration. The endocardium was thickened and subendocardial tissues showed fibrosis and slight leucocytic infiltration.

CASE 59—(16-173)

A married woman, aged 32, was admitted with the complaint of attacks of shortness of

breath, with blueblackness of the lips and nail-beds, on exertion.

Her FAMILY HISTORY was irrelevant except for the fact that a sister with whom the patient had been associated, had died of a fulminating pulmonary tuberculosis.

In her PAST HISTORY she had had typhoid fever at the age of 16 and mastoiditis at 18. At the age of 20 years, she was confined to a hospital for about a month with an attack of pleurisy in the left chest. After this, for some years, she frequently had pain in this side. She was troubled with a productive cough in the winter.

At 22, while playing tennis, she was seized with a sudden pain in the side and acute shortness of breath. She coughed violently and expectorated a tablespoonful of blood. Ever after this, severe exertion brought on these same symptoms. Gradually less and less exertion was required to cause the attack. After seven years, even the very modified type of tennis and, in fact, even moderately brisk walking (especially upstairs) could not be indulged in. The symptoms were exaggerated following a course of salvarsan. Even the exertion of sweeping would cause violent shortness of breath, a harsh cough, bloody sputum, and a conspicuous blue-blackness of the nail-beds and lips. Rest relieved the symptoms.

Her PRESENT ILLNESS was an exaggeration of her lung trouble. She contracted a severe cold and the acute bronchitis was accompanied by asthmatic paroxysms which were relieved by adrenalin given intramuscularly. However, a severe attack came on and would not respond to the usual measures and, consequently, she was brought to the hospital.

The PHYSICAL EXAMINATION on admission showed an orthopneic, cyanotic woman, apparently IN EXTREMIS, with a harsh rasping cough. Her temperature was normal and the pulse rate was 125 per minute. The neck veins were slightly engorged. The accessory respiratory muscles were brought into action. The left side of the chest did not move with the respirations. The percussion note was tympanitic over the left side of the chest and hyperresonant over the right. The breath sounds on the left were distant and tubular. The breath sounds on the right were puerile. The heart dulness was obscured, the heart was displaced to the right. The sounds were muffled, but no abnormality was noted. The left pneumothorax was relieved by the aspiration of air and the symptoms subsided considerably.

The lungs, during the attack, showing puerile breathing and wheezing rales, while after the attack the expiratory murmur was exaggerated, but no rales were present. The percussion note was still hyperresonant.

The heart was not definitely enlarged and the dulness extended 4.5 cm. to the right and 10 cm. to the left of the midsternal line. The aortic dulness measured 6 cm. The blood pressure was 135/90 to 115/75. The brachials and radials were soft and pliable.

The abdomen was soft. The liver, spleen, and kidneys were not palpable.

The extremities were negative and the reflexes were normal.

After the attack, the cyanosis entirely disappeared, the pink color returned to the mucous membranes, and the patient was entirely relieved and comfortable.

LABORATORY DATA: The blood showed 95 per cent hemoglobin; 4,850,000 red cells, and 6,500 white cells per cu. mm. The differential count showed 2 per cent eosinophiles, but was otherwise normal. The Wassermann was negative. The tuberculosis complement fixation test was positive.

The urine was acid in reaction and contained no albumin, casts or blood cells.

The sputum contained blood, but no tubercle bacilli.

The X-ray plates of the chest showed emphysema and calcified hilus glands.

The electrocardiogram showed questionable right ventricular preponderance and occasionally extrasystoles.

COURSE IN THE HOSPITAL: The symptoms were produced by less and less exertion. Respiratory infections increased her difficulties and as a result she steadily lost ground and died about two and a half years after coming under observation; that is, about twelve and a half years after the beginning of her trouble.

The CLINICAL DIAGNOSES were: Extreme pulmonary emphysema, left pneumothorax (transient), chronic pulmonary tuberculosis, acute and chronic bronchitis with asthmatic attacks. Hemorrhoids.

The PATHOLOGICAL DIAGNOSES were: Bilateral chronic alveolar emphysema, bilateral pulmonary congestion. Sarcoma of the left kidney with metastases along the left ureter; focal necrosis in the liver, acute parenchymatous nephritis in the right kidney. Focal calcified tuberculous nodules in both lungs and regional lymph glands, focal fibrous pleurisy at the left apex. Adenoma of the left adrenal with hemorrhage into the medulla.

THE GROSS AND MICROSCOPICALLY DESCRIPTIONS.

KIDNEYS: The left kidney was about three times the size of the right, but the lower two-thirds of the left kidney was made up of sarcomatous tissue. The right kidney was normal in size, shape and position. The capsule stripped without difficulty, exposing a pale red smooth surface. The cut surface showed normal markings and one yellowish-gray focus 1 mm. in diameter.

Microscopically, the left kidney showed a vascular, large spindle-celled sarcoma. The right kidney showed advanced degeneration of the tubular epithelium, amounting in places to necrosis of foci of considerable size. Tube casts were frequently seen both in the convoluted and collecting tubules. The glomeruli seemed quite large. The blood vessels were markedly congested.

The aorta and pulmonary artery appeared normal.

The HEART was not enlarged. The valves appeared normal in every way. The leaflets were thin and the valve rings were not dilated.

Microscopically, the epicardium was only slightly thickened, while the subepicardial fat was increased. The myocardial cells were hypertrophied and fragmented. The endocardium was thickened to a slight degree.

CASE 60—(68-A)

A man, aged 40, was admitted to the University of Michigan hospital with the complaint of shortness of breath, cough and "dropsy".

PAST HISTORY: He had had an attack of acute rheumatic fever in childhood, gonorrhea in 1907, pneumonia in 1915 and influenza in 1920.

The **FAMILY HISTORY** and the **MARITAL HISTORY** were irrelevant.

PRESENT ILLNESS: The attack of influenza about two years before admission had apparently precipitated all of his symptoms. During this acute illness which had confined him to his bed for almost two months, he had had a severe cough, shortness of breath and a dull pain over the precordium. He had not only never been free from the symptoms following their onset but had noted a gradual but definite increase in the severity of the same. He had not been able to lie on his left side for two years; and for ten months he had not been able to lie flat in bed; and for three months he had not been able to recline at all. Palpitation and oedema had been troublesome for about nine months and had also increased in severity. Expectoration of frothy brown sputum in considerable amounts had been present for months and hemoptysis had been noted for about a week. The patient had lost 50 pounds of weight within a year.

PHYSICAL EXAMINATION: The patient was orthopnoeic, emaciated, distinctly pale and haggard. The conjunctivae had a distinct subicteric tint. The mucous membranes were pale and cyanotic.

The neck veins were moderately engorged. The thorax was fairly symmetrical anteriorly, while posteriorly there was a marked scoliosis.

The cardiac impulse was in the sixth interspace, 14 cm. to the left of the median line. A long diastolic thrill was palpable over the area of the maximum cardiac impulse. A distinct diastolic shock was palpable in the pulmonary area. On auscultation a long rumbling diastolic murmur and a blowing systolic murmur were heard over the cardiac impulse. The second sound in the pulmonary area was very loud, while the aortic second sound was weak. The rhythm was regular. The blood pressure was 100/80.

The examination of the lungs revealed the signs of fluid at the left base and moisture throughout both lungs, and a pleural friction rub throughout the right chest.

The abdomen was full. The liver was considerably enlarged and tender. The spleen was not felt. There was a question of the presence of a small ascites.

There was a massive oedema of the extremities. The reflexes were diminished but otherwise normal.

THE LABORATORY EXAMINATIONS

The urine contained albumin and numerous hyalin and granular casts and leucocytes.

The blood examination revealed a slight secondary anemia. The differential count was normal. The Wassermann reaction was negative.

The pleural fluid, 1300 c.c. of which was removed from the left chest, was reddish brown, of a specific gravity of 1.012 and contained 15,000 red and white cells per c. mm.

The electrocardiograms showed marked right ventricular preponderance and prominent notched P waves.

COURSE IN THE HOSPITAL: The patient lost some oedema on restricted fluid intake but suffered a great deal from thirst. Digitalis tincture was given to the point of producing heart block without getting clinical improvement. The pleurisy on the right side became more extensive, the temperature rose to 100° occasionally. The patient became irrational, lost ground rather rapidly and died twenty days after admission.

The **CLINICAL DIAGNOSES** were: Rheumatic heart disease. Mitral stenosis and insufficiency. Cardiac failure with congestion and oedema. Acute fibrinous pleurisy on the right. Chronic pulmonary tuberculosis.

The **PATHOLOGICAL DIAGNOSES** were: Chronic mitral endocarditis. "Button hole" stenosis with insufficiency. Cardiac hypertrophy and dilatation, (rheumatic (?) endocarditis and myocarditis, and streptococic (?) myocarditis and pericarditis). Brown induration of lungs with multiple hemorrhagic infarctions in all stages, emboli and thrombi in all stages. Purulent broncho-pneumonia. Organizing fibrino-purulent pleuritis on the right side. Right sided atelectasis and artificial pneumothorax. Chronic adhesive pleuritis on entire left. Marked nutmeg liver with early cirrhosis. Extreme passive congestion of all organs. Multiple anemic infarctions of spleen and kidneys. Chronic stasis catarrhal gastro-enteritis. Chronic cystitis. Advanced atherosclerosis of aorta and pulmonary arteries. Fatty atrophy of pancreas with hypertrophic islands of Langerhans. Lipoidosis of adrenals. Aspermatogenesis. Atrophy and edema of all fat tissues.

THE GROSS AND MICROSCOPICAL DESCRIPTIONS.

KIDNEYS: The left kidney weighed 235 grams and measured 14x6½x4 cm. The fatty capsule was very scant. The fibrous capsule stripped very readily. On section the kidney bled freely. There were a few small depressed areas like old infarction. The pelvic mucosa was negative. The right kidney weighed 230 grams and measured 12½x6½x4 cm. On section it showed more marked congestion than was found on the right. In addition to the small depressed areas seen on the left there was one large, irregular depression which was healed. The cut surface also showed one small hyalin area which was apparently a tubercle.

Microscopically, the kidneys showed marked passive congestion. There were hyalin casts in some of the collecting tubules, but no true nephritic changes. The renal arterioles contained numerous hyalin emboli. There were also organized emboli and small healed infarcts in the cortex.

AORTA: The thoracic aorta and the abdominal aorta showed respectively moderate and marked atherosclerosis.

Microscopically the picture was that of an advanced non-syphilitic aortitis or arteriosclerosis.

HEART: The pericardial sac was free of adhesions and contained about 120 c.c. of clear slightly icteric fluid. The heart measured $18 \times 11\frac{1}{2} \times 10\frac{1}{2}$ cm. and weighed 745 grams with the vessels cut short and the blood washed out. The auricles weighed 165 grams and the ventricles 580 grams. The subepicardial fat was much reduced in amount. The epicardium showed two well defined soldier's spots over the anterior surface of the right ventricle. The apex was in the 6th interspace midway between the anterior and middle axillary lines. The right border was $5\frac{1}{2}$ cm. to the right of the midline. The heart lay somewhat transversely in the pericardial sac and had rotated slightly so that only the right-sided structures presented anteriorly. Neither the left auricle nor the left ventricle could be outlined at all from the anterior aspect. The apex was definitely bifid being formed more by the right ventricle than by the left. On the posterior surface there were numerous subserous hemorrhages especially along the auriculo-ventricular groove. The tricuspid orifice admitted three fingers with ease, four with difficulty; the pulmonary admitted two fingers with ease and would not take three; the aortic admitted the thumb with ease. The mitral orifice was reduced by thickened calcified leaflets to a typical button slit 2 cm. long and 2 to 4 mm. wide. About three fourths of the mitral ring was the seat of heavy calcareous deposits, which were 1 cm. in thickness in some places. Along the margins of the orifice were still a few very small translucent nodules, apparently relatively fresh vegetations. The valve rings measured: mitral, 11 cm.; aortic 9 cm.; tricuspid, 12 cm.; and pulmonary 9.5 cm. The pulmonary cusps were considerably fenestrated at the attachments at the upper borders.

Microscopically, there was a generalized marked atrophy of the heart muscle with, however, a goodly number of hypertrophic fibers scattered about and many areas showing slight fatty degenerative infiltration especially near the endocardium. The larger coronary arteries showed very little sclerosis, but there were localized patches of fibrosis around the vessels. There was no active reaction and no Aschoff cells. A chronic endocarditis still slightly active but without calcification was present on the valve sections. The pericardium showed a nearly healed pericarditis with sclerosis replacing the subepicardial fat with some slight infiltration. Some bands of fibrosis showing infiltration extended from the epicardium into the myocardium. This pointed more to a more diffuse myocarditis more like a streptococcus endocarditis rather than the healed rheumatic type that the rest of the picture resembled.

